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THE NORMAL HEART AND CONDITIONS SIMULATING CARDIAC DISEASE.[1]

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THE importance of organic diseases of the heart as a leading cause of morbidity and mortality in the community needs no emphasis. Vital statistics lend support to the following picturesque statement of Boyd:[(2)] "Of all the ailments which may blow out life's little candle, heart disease is the chief."

Furthermore, the well-known fear of heart disease exhibited by the laity, an idea usually associated with sudden and unforeseen death, should stimulate us in our endeavours to differentiate the normal heart and conditions simulating cardiac disease from morbid states associated with irreversible structural damage to the heart. We should remember that practically every symptom suggestive of cardiac disease may occur in the absence of any detectable abnormality of the heart, and thus before committing ourselves to a positive diagnosis of a cardiac lesion we should be very sure of our ground. The harm done by a mistaken diagnosis is difficult to remedy and may prove a handicap to the patient throughout his life. Moreover, since the commencement of the war, the examination of thousands of men and women in order to determine their fitness for entrance to the various Services has invested the subject of the normal heart with a new and national importance.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 28, 1942.

DATA.

As the time available for the preparation of this paper was limited, I was unable to examine in detail a large number of normal subjects. I therefore decided to examine 15 students from my class at the Sydney Hospital, and I selected 45 out of the last 2,000 new patients seen in private practice to provide examples of conditions simulating cardiac disease. The cases have been classified in the following way.

Normal medical students		15
Conditions simulating cardiac disease—		
Anæmia	1	
Emphysema	1	
Panniculitis	1	
Fibrositis	4	
Muscle sprain	2	
Pleurisy	1	
Pain of gastric distension	2	
Neuro-circulatory asthenia	33	
	—	45
Total		60

Details of the cases are summarized in Tables I and II.

THE NORMAL HEART.

It is a matter of some difficulty to present a satisfactory definition of the normal heart. King makes the following statement:

The heart may be considered normal (L. *normalis* from *norma*, rule or pattern) provided it conforms both in function and structure with the pattern determined from the examination of the hearts of healthy individuals.

In the determination of the normality of the heart, the clinician should inquire into the patient's history (with special reference to his occupation, habits and bodily

TABLE I.

Findings in Fifteen Normal Medical Students.[1]

Case No.	Sex.	Age. Yrs.	Weight.	Height.	Apex Beat: Distance from Mid-line in Inches. (Subject Recumbent.)	Heart Sounds.	Blood Pressure (Millimetres of Mercury.)		Radial Pulse Rate. (Per Minute.)	X-ray Measurements in Centimetres: Subject Erect.			Electrocardiogram.	Remarks.
							Systolic.	Diastolic.		Transverse.		Cardio-thoracic Ratio.		
										Max. Right.	Max. Left.			
			st. lb.	ft. in.										
I	M.	23	12 0	5 11½	4·0	Normal.	130	92	84	3·9	7·5	2·8	Normal.	Scarlet fever at ten years.
II	M.	22	9 9	5 9½	3·5	Pulmonary systolic impurity.	130	84	79	4·5	6·0	2·6	Normal.	
III	M.	22	10 12	5 10½	4·0	Normal.	130	84	62	3·5	7·0	2·6	Normal.	
IV	M.	20	10 1	5 9	4·0	Normal.	130	80	94	2·3	9·5	2·4	Normal.	
V	M.	19	11 5	5 8	3·75	Aortic first sound impure, second accentuated.	140	90	85	4·3	7·5	2·5	Normal.	Suffers from occasional palpitation.
VI	M.	22	12 5	6 1	4·0	Pulmonary second sound accentuated.	130	82	66	3·7	9·7	2·3	Normal.	
VII	M.	21	11 7	6 1	4·0	Normal.	130	80	84	3·7	8·9	2·1	Left axis deviation (slight.)	Scarlet fever at 11 years. Father died of endocarditis at 50.
VIII	M.	24	11 12	5 11½	3·5	Normal.	110	86	82	3·0	8·3	2·5	Normal.	
IX	M.	20	12 7	6 2	3·75	Pulmonary second sound accentuated.	134	80	80	4·0	7·0	2·6	Normal.	Bronchopneumonia at three years.
X	M.	21	12 7	6 2	3·75	Normal.	130	80	72	3·6	8·3	2·7	Normal.	
XI	M.	21	9 7	5 8	3·25	Normal.	130	90	66	3·3	8·2	2·3	T' waves in lead II biphasic and in lead III inverted (see Figure I).	Diphtheria at five years.
XII	M.	20	12 7	6 0	4·0	Normal.	120	80	79	4·3	8·5	2·3	Normal.	
XIII	F.	20	7 7½	5 1½	3·5	Second sound accentuated at all areas.	138	90	76	2·5	8·0	2·2	Normal.	Diphtheria at nine years.
XIV	F.	20	11 11	5 10½	4·0	Second sound accentuated at all areas.	130	90	88	4·2	8·0	2·3	Simple tachycardia (rate 110 per minute).	
XV	M.	22	13 8½	5 9	4·25	Pulmonary second sound accentuated.	130	90	84	4·0	9·5	2·3	Normal.	Scarlet fever and acute nephritis at nine years.

[1] Exercise tolerance tests produced normal responses, and retinal arteries appeared normal in every case. Weight and height were measured with the subject clothed.

activities), his previous health and the medical history of his relatives, and should afterwards conduct a general routine physical examination. During the investigations the examiner should note the patient's psychological reactions and his psychological background. In certain cases X-ray and electrocardiographic studies may be invaluable aids in the elucidation of the problem.

Such complaints as breathlessness, especially breathlessness which is increased on exertion, precordial pain (if substernal in situation) and palpitation may be suggestive of organic cardiac disease.

A history of syphilitic infection or of previous attacks of rheumatic fever would draw attention to the possibility of the presence of syphilitic aortitis or rheumatic valvular disease. The occurrence of cerebral vascular catastrophes or of coronary artery disease or of essential hypertension among other members of the patient's family should make the physician consider carefully the likelihood of degenerative cardiac disease in his patient.

In the differentiation of the normal heart from conditions associated with organic cardiac disease, an inquiry as to the age of the patient will furnish important information.[2] Clinical experience has shown us that congenital cardiac abnormalities are usually first noticed in childhood, while rheumatic heart disease is generally established before the end of the second decade, that syphilitic cardiovascular infection is most common in middle life, and that coronary disease and hypertensive disease are encountered most frequently in the fifth, sixth and seventh decades. While exceptions to this grouping are often noted, it is evident that much valuable information may be gleaned from a simple inquiry as to the patient's age.

PHYSICAL EXAMINATION.

Physical examination should be comprehensive; but in this paper only the details of the cardio-vascular examination will be described.

Inspection.

In a person with a normal heart, cyanosis, dyspnœa, orthopnœa and œdema of the dependent parts should be absent. Prominence of the precordium may be present in normal subjects. The position of the apex beat is a most important indication of the size of the heart. It is situated in the fifth intercostal space, usually not more than four inches from the mid-line. In the fifteen normal students the apex beat was found in the fifth intercostal space at distances varying from 3·25 to 4·25 inches.

As Parkinson[3] has stated: "It is remarkable how seldom the apex beat is found beyond its rightful place unless the heart is affected; hence its permanent value in diagnosis." Displacement of the cardiac impulse may be found in scoliosis, in which it results simply from twisting of the thorax, or it may occur because the heart is pulled or rotated from its rightful position by a fibrotic condition of the lung or pushed therefrom by a pleural effusion or a pneumothorax. It is important also to remember the displacement found with congenital dextrocardia.

A diffuse impulse does not necessarily indicate enlargement. In the presence of tachycardia the apex beat may appear to be displaced, but there may be no cardiac enlargement. Reexamination after disappearance of the tachycardia should decide the point. It is as well to remember that in certain cases the tachycardia may be the result of an accompanying febrile affection, such as pulmonary tuberculosis, or that it may be a manifestation of thyreotoxicosis.

Parkinson[3] sums up the position in the following way: "If neither tachycardia nor scoliosis explains an apex beat beyond the nipple line, and the lung is not fibrotic, the sign indicates cardiac enlargement."

Palpation.

Palpation should confirm the position of the cardiac impulse noted by inspection. The apex beat is the lowest

TABLE II.

Conditions Simulating Cardiac Disease.

Case Number.	Sex.	Age. (Yrs.)	Clinical Features.	Blood Pressure. (Millimetres of Mercury.)		Fluoroscopic Findings.	Electrocardiogram.
				Systolic.	Diastolic.		
XVI	F.	43	*Anæmia:* Complained of submammary pain which she thought was caused by heart trouble. Blood examination showed microcytic hypochromic anæmia. Pain disappeared after iron therapy. Also suffered from giddy turns.	130	90	—	—
XVII	M.	52	*Emphysema:* Breathlessness, increased by exertion, of two years' duration. Chest barrel-shaped, breath sounds faint.	130	100	Normal.	Normal.
XVIII	F.	30	*Panniculitis:* Pain just below and medial to apex beat. Small area of superficial tenderness. Easily breathless.	150	100	Normal.	Simple tachycardia (rate 100 per minute).
XIX	F.	54	*Fibrositis:* Pain radiating medially from 7th left intercostal space in mid-axillary line. Easily breathless. Tenderness on deep pressure over 7th left space in mid-axillary line.	140	100	Normal.	Left axis deviation.
XX	F.	18	Pain over left lower intercostal spaces in axillary region of four years' duration. Tender on deep pressure over the same area. Nodules palpable. Breathlessness.	140	90	—	—
XXI	M.	53	Pains in left side of chest radiating to shoulder and elbow of 18 months' duration. More easily breathless than previously. Tender on deep pressure over left deltoid.	144	100	Normal.	Left axis deviation.
XXII	F.	33	Pain in left infrascapular region. Breathlessness. Palpitation.	140	90	Heart smaller than normal.	Left axis deviation.
XXIII	M.	47	*Muscle Sprain:* Pain in region of 3rd, 4th and 5th costochondral junctions after lifting a sheep. Tender over same area. Rheumatic fever at 20 years of age. Apex beat five inches from midline in 6th intercostal space. Aortic and mitral systolic murmurs.	150	80	Widened aorta. Hypertrophied left ventricle. Generalized cardiac enlargement.	Ventricular extrasystoles.
XXIV	M.	54	Pain in left infrascapular region after lifting a can of milk weighing 140 lb. Area of tenderness on palpation just below angle of scapula.	120	90	Normal.	Normal.
XXV	M.	49	*Pleurisy:* Pain in lower part of left side of chest worse on breathing. No friction rub heard.	150	100	Restricted movement of left diaphragm.	Normal.
XXVI	M.	63	*Pain of Gastric Distension:* Pain in lower part of left side of chest radiating down left arm, worse after food. Not easily breathless.	160	100	Slight enlargement of left ventricle and aorta.	Normal.
XXVII	M.	55	"Pain round heart" for many years, worse after food and worry. Not breathless.	130	80	Normal.	Left axis deviation.
XXVIII	M.	29	*Neuro-circulatory Asthenia:* Weakness, dizzy feelings, fainting attacks, constricting sensation in neck. Mitral first sound impure.	140	90	Normal.	Sinus arrhythmia.
XXIX	M.	38	Palpitation. Pain in region of left nipple. Loss of weight. Second sound reduplicated at apex.	164 (January 10, 1940) 130 (January 19, 1940)	100 90	Normal.	Delayed auriculo-ventriculo-conduction due to digitalis 10/1/40. Normal 19/1/40 (see Figures X and XI).
XXX	M.	30	Fainting turn. Giddy turns. Aortic systolic murmur. Aortic second sound accentuated.	150	90	Normal.	Right axis deviation.
XXXI	F.	42	Complains of "a sensation of floating round", "unable to take a deep enough breath". Pain in left sub-mammary region. Breathless.	150	100	Normal.	Normal.
XXXII	F.	29	Palpitation. Notices rapid heart action at times. Submammary pain. Mitral first sound impure.	120	90	Normal.	Normal.
XXXIII	M.	48	Submammary pain radiating to spine. Easily breathless. Collapsing turns. Has suffered from duodenal ulcer.	120	80	Normal.	Left axis deviation.
XXXIV	F.	35	Submammary pain radiating to spine. Easily breathless.	120	80	Normal.	Normal.
XXXV	M.	57	Pulsation in neck. Noticed "pulse irregular at times". Has had much worry. Aortic second sound accentuated.	170	120	Aorta slightly increased in width.	Normal.
XXXVI	M.	30	About two months previously "seemed to wake up and catch his breath". Suffers from breathlessness and lack of concentration. Worried about engagement.	130	90	—	—
XXXVII	M.	54	"Black outs." Submammary pain. Influenza three months previously.	130	80	Normal.	Left axis deviation.
XXXVIII	F.	25	Fainting turns at intervals for ten years, brought on by excitement or exertion. Pain in upper portion of left side of chest and left arm. Easily breathless.	110	80	—	—
XXXIX	M.	32	Pain in upper portion of left side of chest. Not easily breathless. Obese (weight 13 st. 6½ lb., height 5 ft. 9½ in.).	130	80	Normal.	Left axis deviation.
XL	M.	63	Submammary pain ever since return from war 21 years previously. Suffered from "D.A.H."; "lost the use of his left leg and arm" eight days previously. Is easily breathless.	120	80	Aorta sclerotic.	Normal.
XLI	M.	37	Palpitation for three months. Ache in left submammary region.	120	80	Normal.	Normal.
XLII	F.	15	Becomes easily tired. Not allowed to participate in sport because of two attacks of rheumatic fever, one at 2½ and the second at 6 years of age. Musical mitral systolic murmur not conducted.	140	90	Normal.	Normal.
XLIII	M.	42	Noticed pain in left submammary region and irregular cardiac action after a recent attack of influenza.	104	70	Normal.	Left axis deviation.
XLIV	M.	45	Giddiness and fainting turns. Worry.	140	90	—	Normal.
XLV	F.	62	Recent attack of pertussis. Heart beats audible in ears. Left mammary pain.	170	100	Normal.	Normal.
XLVI	M.	25	Collapsing turns. Queer feelings in the head. Pain in left side of chest. Easily breathless.	140	80	Normal.	Sinus arrhythmia.

TABLE II.—*Continued.*

Conditions Simulating Cardiac Disease.—Continued.

Case Number.	Sex.	Age. (Yrs.)	Clinical Features.	Blood Pressure. (Millimetres of Mercury.)		Fluoroscopic Findings.	Electrocardiogram.
				Systolic.	Diastolic.		
XLVII	M.	35	"Collapsed" two years previously. Said to be suffering from "low blood pressure". Easily breathless. Feels "heavy on chest" after smoking a few cigarettes. Palpitation. Wakes up "wanting to take a breath". With excitement he "goes blue at times and white at other times". Tonsils unhealthy.	120	80	——	——
XLVIII	M.	30	Pulse becomes rapid "for no reason". Choking sensations. Submammary pain.	150	90	Normal.	Simple tachycardia (rate 105 per minute); right axis deviation.
XLIX	M.	43	Giddy attacks. Easily breathless. Exhaustion and fatigue. Marital worry.	120	80	——	——
L	F.	65	Giddy attacks. "Nervous heart" for many years. Ache in left side of chest. When overtired unable to sleep "on account of heart beating". Mitral systolic murmur.	160	100	Normal.	Left axis deviation.
LI	F.	29	Tightness in left side of chest during past 12 months, worse on exertion. Easily breathless. Heart sounds forcible.	130	90	Normal.	Normal.
LII	M.	49	Dull ache in left submammary region. Easily breathless. Has suffered from considerable worry.	120	80	Normal.	Normal.
LIII	M.	27	Recent "collapse". Submammary pain. Worry about engagement. Business anxiety.	120	80	Normal.	Normal.
LIV	F.	46	Giddiness. Easily breathless. Tingling of fingers.	134	90	——	——
LV	M.	34	Noticed that heart missed a beat six months previously. Dizzy turns. Easily breathless.	140	100	Normal.	Ventricular extrasystoles.
LVI	F.	27	Dull ache in left mammary region. Easily fatigued. Aortic and mitral systolic impurities. Tonsils unhealthy.	140	100	Normal.	Normal.
LVII	M.	34	"Not fit for years." Feels giddy. Pain in left side of chest. Easily breathless. Tired. Complains of "sighing breathing". Orthostatic albuminuria.	140	100	Slight enlargement of right ventricle.	Normal.
LVIII	F.	17	"Hot flushes" and giddiness for six months. Throbbing in head, frontal and occipital. Attacks of breathlessness. Fainting turns. Palpitation.	136	90	Normal.	Normal.
LIX	M.	48	Giddiness, discomfort in region of heart. Easily breathless. Feels "hazy in the head and uncertain of himself". Sweating in palms of hands.	140	90	——	——
LX	M.	32	"Cramp in left side of chest." Run down. Heavy smoker (50 to 60 cigarettes daily). Had been under considerable nervous strain.	146	90	Heart smaller than normal.	Normal.

and outermost point at which the examining finger is distinctly raised by each beat of the heart. The apex beat may not be palpable when the chest wall is thick or when emphysema is present, or more rarely, when the heart is acting feebly. Under the influence of excitement a fine vibratory sensation may be transmitted to the examining hand; but this differs in both time and intensity from the presystolic thrill of mitral stenosis, which "has been aptly compared to the sensation produced when the hand is placed on the back of a purring cat".[4]

Percussion.

In adults the normal area of cardiac dulness should not extend more than four inches to the left or more than 1·75 inches to the right of the mid-sternal line; but there may be a slight increase of these measurements in subjects whose hearts lie more than ordinarily in the transverse position. Hamilton[5] studied certain physiological peculiarities of the heart in infancy and childhood, and pointed out that the left ventricle, which is about equal in size to the right ventricle at birth, becomes approximately twice the size of the right ventricle at the end of the first six years of life. Percussion often gives fallacious results. To outline by percussion the hearts of persons suffering from obesity or emphysema is always a matter of extreme difficulty and sometimes an impossibility. Finally, it is always wise to percuss the upper portion of the sternum over the aorta, to exclude the possibility of aortic dilatation or aneurysm.

Auscultation.

As there are many variations of the normal heart sounds, and as the interpretation of physical signs may be of paramount importance in the detection of slight or early heart disease, especially in men of military age, auscultation which is full of pitfalls presents special difficulties.

The heart sounds are usually two in number, represented by the syllables "lub" for the first and "dupp" for the second. A third heart sound has been found in a large proportion of healthy young adults. If it is pronounced, the third heart sound may be mistaken for an early diastolic rumble of mitral stenosis, or for the so-called opening click of mitral stenosis, or for the reduplication of the second sound due to asynchronous closure of the aortic and pulmonary valves. Usually, however, asynchronous closure of these valves causes a reduplication of the second sound best heard at the pulmonary area, while the normal third sound is most prominent at the apex or over the lower part of the precordium. Furthermore, the interval between the two elements of a split second sound is not so prolonged as that between the normal second and third sounds. The third heart sound and the protodiastolic gallop cannot be distinguished by auscultation alone. Indeed, it would appear likely that they are produced by a similar mechanism—namely, the sudden tension of the ventricular walls at the end of filling early in diastole. As a rule the third heart sound is heard in youth and may be regarded as normal, whereas the protodiastolic gallop is found in association with the other signs of serious heart disease. The first sound at the apex may be exaggerated as a result of nervousness, fever or exertion, and normally it is louder in young people than in older subjects. The pulmonary second sound is usually louder than the aortic sound before twenty years of age, but after thirty years the aortic second sound is usually the louder.

Reduplication of either the first or second sound may occur in health. Reduplication of the first sound is uncommon; it has been attributed to the impact of the heart against the chest wall, and is said to disappear when the patient assumes the recumbent position. Such reduplication differs from presystolic gallop rhythm in three respects—namely, its variability with change of position, the relative softness of the first element in presystolic gallop rhythm, and the fact that the latter condition is sometimes associated with bundle branch block. Reduplication of the second sound is not uncommon in health, especially during deep respiration.

Murmurs are frequently heard in subjects with normal hearts. An apical systolic murmur, which is heard when

the patient is in the recumbent position, but which disappears when he assumes the erect posture, apparently has no pathological significance, although some authorities have raised the question as to whether it may be due to a slight degree of mitral incompetence which is not of serious import. Similarly, a basal systolic murmur, if soft and obvious only during recumbency, may generally be ignored, more especially if it is heard only at the pulmonary area ("the area of auscultatory romance").

The so-called cardio-respiratory murmurs are frequently mistaken for evidence of organic disease. They are usually heard only on inspiration and may be found at both the apex and the base of the heart. They are said to be caused by impingement of the heart as it moves in systole against the lung, causing a systolic accentuation of the inspiratory breath sound.

A harsh, apical murmur constantly heard in both the erect and the supine positions and unaffected by respiration, is always suggestive of an organic lesion. Owing to its localization to a small area just medial to the apex beat, the presystolic murmur is often missed. Should there be any doubt, the best procedure is to ask the subject to exercise his arms briskly and afterwards to lay him down on his left side. If it is present, the typical presystolic crescendo murmur will then be heard. The presence of a loud pulmonary second sound will be additional evidence suggestive of mitral stenosis.

Anæmia is a fruitful source of systolic murmurs, both apical and basal. The bruits due to congenital heart disease may be mistaken for functional murmurs. A loud, hissing systolic murmur in the third and fourth left intercostal spaces near the sternum is indicative of a ventricular septal defect. A systolic-diastolic continuous murmur (machinery-like murmur) is characteristic of a patent *ductus arteriosus*.[1]

X-Ray Examination of the Heart.

The common methods of X-ray examination of the heart are fluoroscopy, orthodiagraphy and the examination of the film taken at a distance of six feet.

The variations in the size of the heart at different age periods must be borne in mind. For example, according to Hamilton[5] the right auricle undergoes rapid hypertrophy during the first six weeks of life. Approximately one-third of the heart lies to the right of the mid-line in childhood. The transverse diameter of the heart is approximately one-half of the width of the chest, and there is no change in this relationship with increasing age or height.

In obese subjects, or as a result of such conditions as ascites or abdominal tumour, the diaphragm is usually rather high; this causes the heart to assume a relatively transverse position.

Fluoroscopy is a valuable method of determining the size and shape of the heart and the pulsations of the various chambers. The heart should be examined in the antero-posterior, the right oblique and the left oblique views. Orthodiagraphy is useful when a permanent record of the cardiac silhouette is required.

The size of the heart can be gauged by fluoroscopy, or the diameters of the heart can be measured on the six-foot film. Two diameters of the heart are of great importance—namely, the transverse and the longitudinal diameters. The transverse diameter is the measure of the greatest distance across the heart, and is the sum of two perpendiculars drawn to the mid-line, one from the most prominent part of the right border and the other from the most prominent part of the left border. The longitudinal diameter is measured from the notch which separates the right auricle from the superior *vena cava* to the apex.

The presence of scoliosis may be a cause of confusion. By turning the patient a little to one side, usually the right, one can correct the distortion caused by the spinal curvature.

In the estimation of the size of the heart the cardio-thoracic ratio is of great value. It is the ratio of the transverse diameter of the heart to the internal diameter of the chest. East and Bain state that the width of the heart should not exceed one-half of that of the chest (cardio-thoracic ratio, 1:2). In subjects with long, narrow chests and "hanging" hearts the ratio may be as much as 1:4, whereas in broad, thick-set people whose hearts lie transversely, the ratio may be as low as 1:1·9. Complicated formulæ have been devised in order to eliminate errors due to the different positions of normal hearts. Hodges and Eyster[6] established such a formula to estimate the predicted transverse diameter of the heart from the weight, height and age of the subject.

In the series of normal students the transverse diameters of the heart varied from 10·5 centimetres to 13·5 centimetres and the cardio-thoracic ratio lay between 2·1 and 2·8.

Electrocardiography.

Electrocardiograms from subjects with normal hearts are not infrequently misinterpreted as abnormal tracings. It is proposed to refer to some common sources of error, arising from the effects of posture and of respiration, from the administration of digitalis and from artefacts originating from various causes.

The effect of change in body position and of respiration on the electrocardiogram has been studied in detail by Master.[7] The form of the electrocardiogram may be altered by a change in the position of the heart, the commonest causes of which are alteration in the body position and respiration acting through the movements of the diaphragm. Other conditions such as ascites and obesity may produce the same effect. Master makes the following statement:

> It is evident that rotation of the heart rather than lateral movement is the significant factor in the alteration of the electrocardiographic complexes. The changes in the electrocardiogram may be marked, including distinct axis deviation and even the appearance of a large *Q*-wave in the third lead, and alterations in the *T*-wave. Since the latter are almost universally interpreted as evidence of damage of the heart muscle, it is of great practical importance to recognize that they often represent nothing more than a change in the position of the heart.

In this connexion the conclusions of White, Chamberlain and Graybiel[8] are of great interest. They make the following statements:

> Inversion of the T waves in lead II of the electrocardiogram, although most commonly the result of heart disease or toxic states, may be a normal physiological variation in occasional persons, particularly those of asthenic habitus with vertical hearts and prone to neurocirculatory asthenia.
>
> The position of the heart is the most important factor in producing this T wave inversion which is found in the sitting or standing position but is corrected by recumbency or by elevating the diaphragm as at full expiration. Autonomic nervous influences comprise another factor, although less striking as a rule, the low or inverted T waves then being attended by tachycardia; any cause of such stimulation, for example, excitement can then be responsible. Fear and anxiety may act through the production of overventilation with resultant alkalosis. Both heart position and nervous influences may be active in the same case.
>
> The relatively common occurrence normally of inversion of the T waves in lead II makes it imperative to recognize its existence in order to avoid erroneous diagnoses of heart disease.

In the tracing from one of the 15 normal students (Case XI) a young man, aged twenty-one years, of somewhat asthenic habitus, it was seen that the *T* waves in the second lead were biphasic and in the third lead inverted (see Figure I). This occasioned him great anxiety, as he became doubtful of the integrity of his heart, although he used to ride a bicycle from 70 to 100 miles each Sunday without distress. A second electrocardiogram taken in the recumbent position showed the *T* waves in the second lead to be upright and in the third lead to be biphasic (see Figure II).

It was then decided to take electrocardiograms with the subject in the sitting position and to note the effect of respiration. Upon deep inspiration the *T* waves in the second and third leads were inverted (see Figure III),

[1] At this stage a sound record was played demonstrating the normal heart sounds, murmurs produced by rheumatic valvular disease and murmurs found in congenital heart disease.

while during expiration the *T* waves in those leads became upright (see Figure IV). X-ray films taken with the subject in the standing position during quiet respiration, after deep inspiration (see Figure V) and after expiration

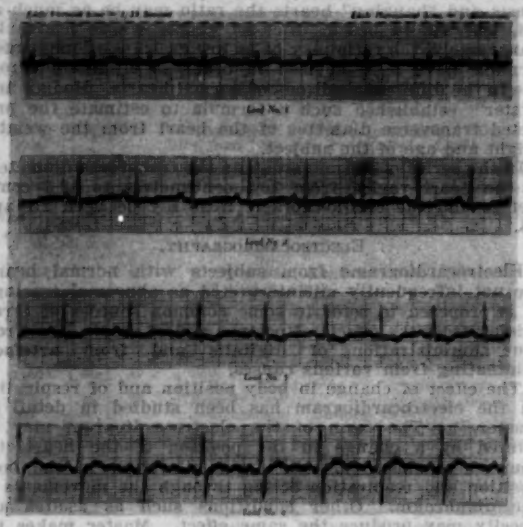

FIGURE I.
Electrocardiogram (Case XI) taken in sitting position, showing biphasic *T* waves in second lead and inverted *T* waves in third lead.

(see Figure VI), and in the recumbent position after deep inspiration (see Figure VII) and after expiration (see Figure VIII), illustrate the variations in the size of the heart shadow resulting from respiration.

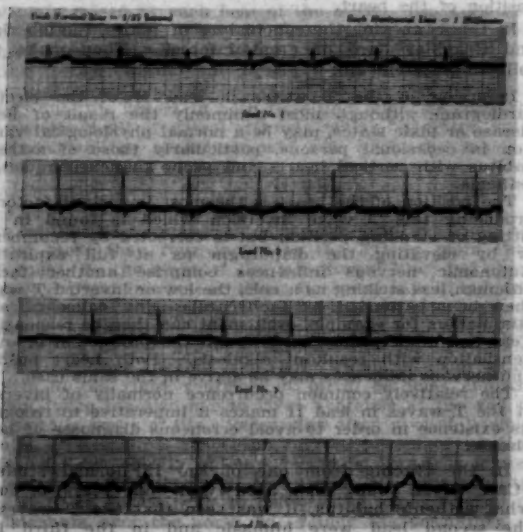

FIGURE II.
Electrocardiogram (Case XI) taken in recumbent position, showing upright *T* waves in second lead and biphasic *T* waves in third lead.

In this connexion the flattening of the *T* waves found in the Yogis by Laubry and Brosse[7] during a "trance" (accompanied by apnœa) are of interest. After the "trance" the Yogi gained "control of his heart" and the electrocardiogram became normal.

In view of the observations of Master,[5] and of White, Chamberlain and Graybiel,[6] alteration in the position of the diaphragm appears to be a likely explanation of the *T* wave changes.

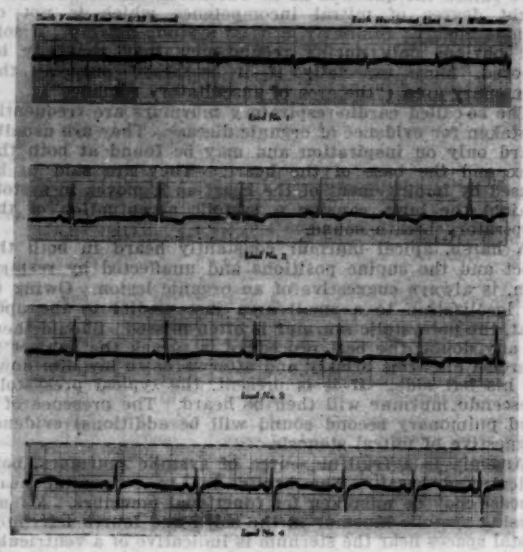

FIGURE III.
Electrocardiogram (Case XI) taken in sitting position after deep inspiration, showing inverted *T* waves in second and third leads.

The results of the administration of digitalis are a frequent source of error. The commonest effect is a depression of the *ST* interval from the iso-electric line

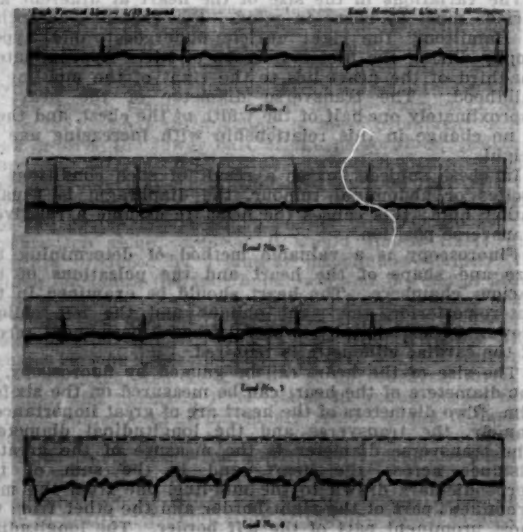

FIGURE IV.
Electrocardiogram (Case XI) taken in sitting position after expiration, showing upright *T* waves in second and third leads.

(the so-called "foot pedal deformity"). This depression may so closely simulate the effect of coronary disease as to make it difficult to determine the causative agent (see Figure IX). Digitalis produces other effects on the electrocardiogram, including varying degrees of heart block. The

patient (Case XXIX) from whom the electrocardiogram shown in Figure X was taken had been given digitalis in the mistaken idea that he was suffering from heart disease. As can be seen, the *PR* intervals are considerably increased and suggest the presence of heart block. After the cessation of digitalis therapy the *PR* intervals returned to normal (see Figure XI).

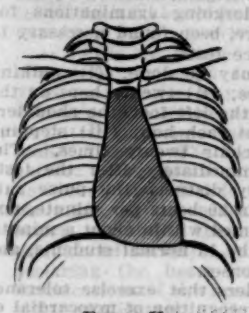

FIGURE V.
Drawing of X-ray film (Case XI) taken with the subject in standing position after deep inspiration. Transverse diameter 10·8 centimetres.

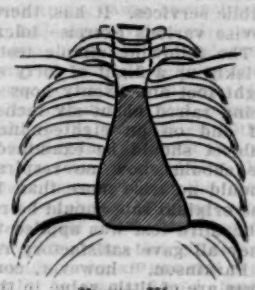

FIGURE VI.
Drawing of X-ray film (Case XI) taken with the subject in standing position after expiration. Transverse diameter 12 centimetres.

Various artefacts may occur as a result of extra-cardiac causes, such as muscular tremor, electrical interference, battery and cable defects *et cetera*. These should not be mistaken for evidence of myocardial disease.

At this stage it will be germane to discuss briefly the place of the electrocardiogram in clinical practice. Obviously it is not a substitute for a careful history and a complete physical examination. As Katz[29] has stated:

> It is only one of the objective approaches to an evaluation of the cardiac state, and is just as subject to personal bias as any other part of the clinical diagnosis. It must be correlated with the rest of the patient's story and findings, and this correlation must be made by the clinician who has seen the patient.

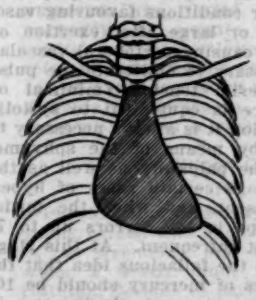

FIGURE VII.
Drawing of X-ray film (Case XI) taken with the subject in recumbent position after deep inspiration. Transverse diameter 13·3 centimetres.

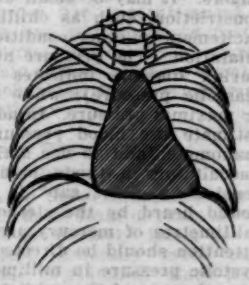

FIGURE VIII.
Drawing of X-ray film (Case XI) taken with the subject in recumbent position after expiration. Transverse diameter 13·5 centimetres.

Age is an important factor in the determination of the normal electrocardiographic standards, for since certain changes may occur as a result of the normal ageing process, what is normal at the age of sixty years may be abnormal at thirty, and the normal record of the infant differs from that of the adolescent or the adult.

The electrocardiogram is invaluable in the elucidation of the arrhythmias. It helps us to distinguish the relatively benign disorders of rhythm, such as sinus arrhythmia or premature contractions, from those of a more serious nature, such as auricular fibrillation or heart block. It may be of value in determining the presence of a right or left ventricular strain. The greatest field of usefulness of the electrocardiogram, however, lies in the management of coronary artery disease. In this condition serial tracings are invaluable in gauging the progress and the prognosis of the lesion, and this information cannot be supplied by any other form of examination.

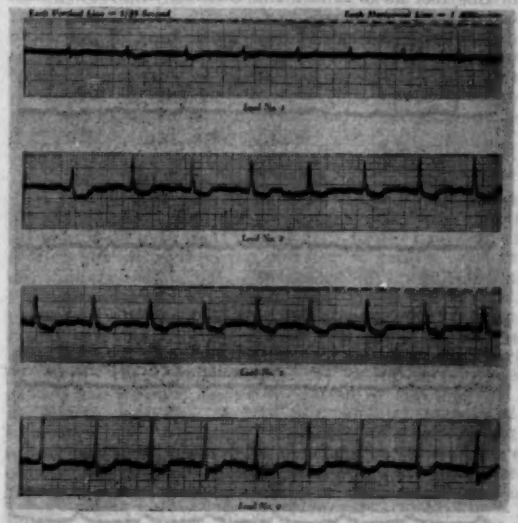

FIGURE IX.
Electrocardiogram showing effects of digitalis. Depressed *ST* interval ("foot pedal deformity").

Finally, it should be remembered that normal variations in the electrocardiogram are numerous, and that a normal electrocardiogram may be found in cases of serious heart disease. The last observation applies especially to the

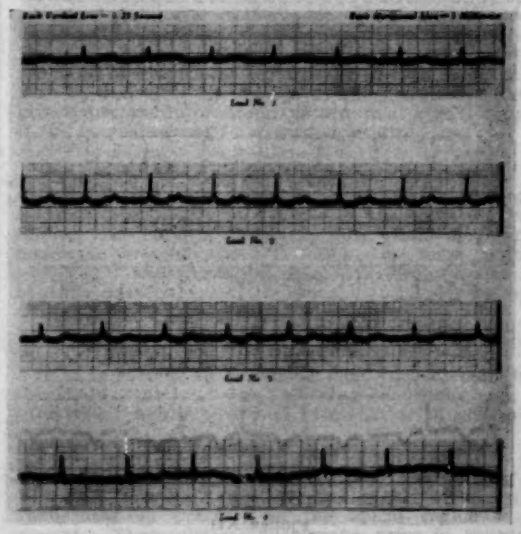

FIGURE X.
Electrocardiogram (Case XXIX) showing increased auriculo-ventricular conduction caused by digitalis.

cases in which only the first three classical leads are taken, and is well exemplified in Figure XII, an electrocardiogram taken from a patient about a month after the occurrence of a coronary occlusion. The first three leads are not very abnormal, but the deep inversion of the *T* wave in lead IV F gives the clue to the diagnosis.

Katz sums up the position in the following way:

In short, the electrocardiograph is not a tool for the unscrupulous or a plaything for the erudite, nor is it an instrument of precision which replaces the ordinary clinical examination. It is as much of an error to expect too much from this tool as to ignore the valuable information that it can give.

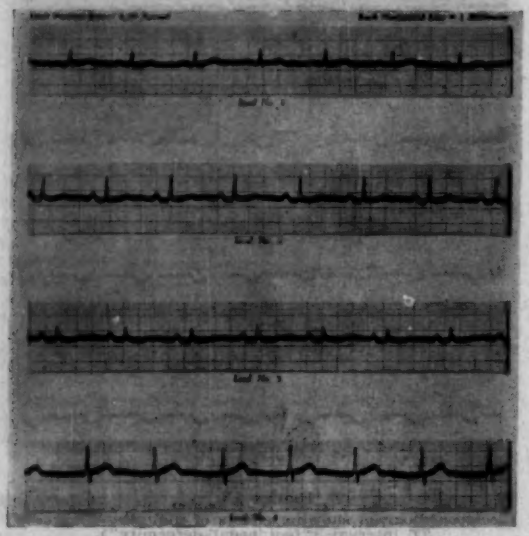

FIGURE XI.

Electrocardiogram (Case XXIX) showing normal auriculo-ventricular conduction after cessation of digitalis therapy.

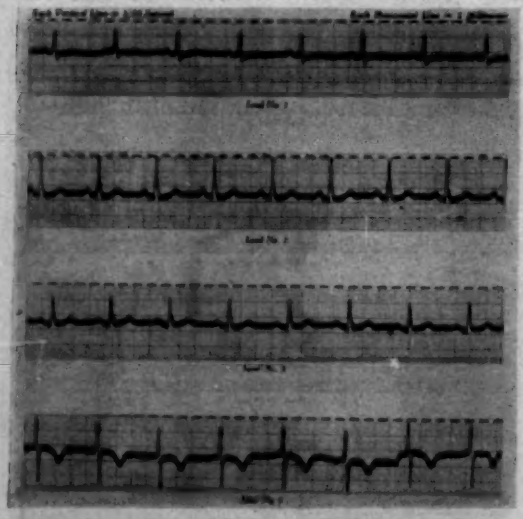

FIGURE XII.

Electrocardiogram taken about a month after the occurrence of a coronary occlusion, showing deep inversion of T wave in lead IV F. (In all other electrocardiograms the fourth lead is IV R.)

The following frequently quoted sentence of Oliver Wendell Holmes ("The Poet at the Breakfast Table", V, page 126) is applicable to the electrocardiograph: "Science is a first-rate piece of furniture for a man's upper chamber, if he has common sense on the ground-floor."

EXERCISE TOLERANCE TESTS.

As Lewis states: "The first indication of cardiac failure is to be found in a diminished tolerance of exercise". For an intelligent patient who tells us that some act that he had previously undertaken without distress now causes undue breathlessness, it is generally unnecessary to set up deliberate exercise tolerance tests. But as Lewis points out, all our patients are not intelligent, nor do they make accurate statements when undergoing examinations for public services. It has, therefore, been found necessary to devise various exercise tolerance tests.

The following simple tests may be done: (i) walking briskly up a flight of forty steps; (ii) twenty hops on the right foot and twenty hops on the left foot, the shoulders being raised about six inches at each hop; (iii) stepping off and on an eighteen-inch chair twenty times. The subject should be examined immediately after the test; he should show no respiratory distress, the pulse rate should not rise more than 10 to 20 beats per minute, and the original rate should be resumed within about a minute. The third test was applied to the 15 normal students, and they all gave satisfactory responses.

Parkinson,[9] however, considers that exercise tolerance tests are of little value in the recognition of myocardial or valvular disease, but that they are valuable in judging the nervous control of the heart rate. Furthermore, Wood states that figures do not support the claims made for these tests. Sir Adolphe Abrahams informed him "that effort-tolerance tests were hopelessly unreliable when performed on world-famous athletes during a contest, when the emotional factor was to the fore".

THE PULSE.

For purposes of convenience the radial pulse is examined in preference to that of other vessels, and such an examination may give valuable information as to the state of the heart. A comparison of the two radial pulses should always be made. According to the traditional method, the state of the vessel wall, the volume, the tension, the force, the rhythm and the rate should be noted. Atheromatous changes are indicated by increased rigidity of the vessel wall and are generally associated with tortuosity of the artery. By volume is understood the apparent size or amplitude of the pulse wave as it passes the palpating fingers. It may be small under conditions favouring vasoconstriction, such as chilling, or large after exertion or excitement or other conditions causing peripheral vascular dilatation. The pressure necessary to obliterate the pulse during diastole indicates arterial tension (minimal or diastolic pressure), while force is equivalent to systolic or maximal pressure. In addition it is always necessary to estimate the blood pressure by means of the sphygmomanometer and to use both the palpatory as well as the auscultatory methods, because in certain cases of hypertension "a silent gap" is present, and when the initial sound heard by the stethoscope is soft, errors up to 70 millimetres of mercury are not infrequent. At this stage attention should be directed to the fallacious idea that the systolic pressure in millimetres of mercury should be 100 *plus* the age of the subject, because no steady increase in blood pressure with advancing years has been noted in normal persons.[10] As was stressed by Mackenzie,[10] arrhythmia is often found in people with normal hearts; the common type is sinus arrhythmia, which is characterized by acceleration during inspiration and retardation during expiration. An intermittent pulse due to premature contractions (or extrasystoles) is common among normal subjects and may be found after excessive use of tobacco or gastro-intestinal disturbances. Generally speaking, arrhythmias of functional origin tend to disappear or diminish after exercise, while the more serious arrhythmias such as auricular fibrillation are aggravated by exertion. The rate of the normal pulse at rest was estimated by Peterson and Walter[11] and found to be 66 per minute in the case of men and 74 in women. The pulse rates of the students ranged between 62 and 94 per minute.

The condition of the retinal vessels as seen with the ophthalmoscope may give important information with regard to the peripheral circulation.

THE DIAGNOSIS OF THE NORMAL HEART.

The following remarks of Lewis are of great importance:

The most valuable indication we possess of a heart's essential soundness is its power to engage fully in its work without complaint. No patient having normal capacity for bodily exercise has grave heart disease; for bodily exercise calls upon the heart for extra work; it challenges the heart's reserves and, if it can soon exhaust these, it will induce unusual breathlessness; or, in the event of the heart's own blood supply proving inadequate, cause anginal pain.

As Lewis has pointed out, full capacity for work cannot always be estimated from the patient's own statement. Thus examination is necessary to exclude definite cardiac enlargement, definite evidence of valvular disease (such as mitral stenosis or aortic incompetence) and congenital malformations. The heart should be auscultated with the patient in the standing and recumbent positions and when he lies on the left side after exercise. An inquiry should be made as to paroxysmal disorders of the heart's action, and should these also be excluded the heart may be pronounced normal. And we should not be deterred from declaring the heart sound either by the presence of modifications of the heart sounds or by an unusual type of electrocardiogram. It should be remembered that an isolated sign can rarely be regarded as being of much significance. As a rule there is more than one indication of disease. These criteria, which were used on a large scale in deciding the fitness of men for active service during the Great War, were found by Lewis to have proved themselves reliable.

Exceptions do occur, and some people with pronounced valvular lesions are able to perform remarkable feats of endurance. The case of the Marathon runner suffering from aortic incompetence and mitral stenosis, in whom the lesions were said to be mutually compensatory, is a classical example.[10] It should be remembered, however, that bacterial endocarditis is frequently engrafted upon chronic valvular lesions.

CONDITIONS SIMULATING CARDIAC DISEASE.

It is not proposed to deal with acute emergencies or with any diseases in which cyanosis, orthopnœa and œdema of the dependent parts are presenting signs.

The conditions which may simulate cardiac disease are numerous and varied. The three principal symptoms suggestive of organic disorder are breathlessness, precordial pain and palpitation, and it is proposed firstly to refer to some morbid conditions causing these symptoms, and secondly to discuss certain aspects of neuro-circulatory asthenia, a disease in which these three symptoms are frequently found in combination.

Conditions Causing Breathlessness.

Physical Unfitness.

Physical unfitness may cause undue breathlessness in a young person who undertakes some form of exercise which is too strenuous, and it is particularly liable to occur when a weedy, unfit subject, generally much out of training, decides to participate in athletics. Such persons frequently suffer from the effects of focal sepsis and from anæmia.

Anæmia.

Unsuspected anæmia is not infrequently the cause of dyspnœa, which may be mistaken for cardiac disease. At times in older subjects the breathlessness is associated with precordial pain. The colour of the patient is generally quite good and the cause of the breathlessness is not obvious until the result of the blood count is obtained.

Some years ago I saw a patient suffering from breathlessness and precordial pain. No abnormality was found on investigation of the circulatory and respiratory systems; but a blood count revealed a hypochromic microcytic anæmia which was found to be due to bleeding hæmorrhoids. After attention to the hæmorrhoids and the administration of iron the symptoms disappeared and the patient has remained well.

The case (Case XVI) of a woman suffering from idiopathic hypochromic anæmia is recorded in Table II. In some cases the anæmia may be of the Addisonian type. Professor Meakins[11] mentioned such a case in his "Medical Essays". The patient, who was a physician, was an enthusiastic walker and the leader of his walking club. He found that his response to effort was becoming seriously impaired, and he consulted many colleagues, as he was certain that he was suffering from a cardiac lesion. Later respiratory disease was suspected. As no evidence of cardiac or respiratory disease was found he was thought to be neurotic. His colour was fairly good and at rest he looked the picture of health. A careful blood examination revealed a hyperchromic macrocytic anæmia; liver therapy produced a rapid return to normal, and again he became the leader of his walking club.

Obesity.

In the obese person the correct diagnosis of the cardiac condition is often a matter of great difficulty. Shortness of breath is frequently present. Obese persons have a diminished vital capacity, develop a greater oxygen debt on exercise and have increased oxygen consumption. Furthermore, the examination of the heart of the obese person is always difficult. The apex beat may not be easily palpable, and the mitral first sound may appear to be fainter than normal owing to the thickened parieties. While it would appear that the obese person does not always suffer from fatty infiltration of the heart, it must be remembered that obesity is frequently associated with hypertension. East and Bain are of the opinion that atheroma of the coronary arteries is found more frequently in fat than in thin persons. For these reasons the heart should be examined very carefully when any obese person complains of undue breathlessness. X-ray and electrocardiographic studies may be essential in such cases.

Emphysema.

In its advanced stages emphysema is the causative factor of right-sided heart failure in many cases. A mild grade of emphysema may be responsible for a certain amount of shortness of breath, especially in the middle-aged and elderly (see Case XVII). Whether the changes in the lungs themselves or the limitation of movement of the thoracic cage are the cause of the dyspnœa is open to question. Whatever is the cause, exertion easily produces dyspnœa, because the capacity for ventilation is diminished. These symptoms may suggest a cardiac lesion, and as in the case of obesity, assessment of the state of the heart may be difficult. Estimation of the circulatory rate can give important information, as a normal result is unlikely if the breathlessness is of cardiac origin.

Other Respiratory Diseases.

Pulmonary fibrosis sometimes causes breathlessness and is not infrequently associated with signs of cardiac failure. In latent pulmonary tuberculosis or pulmonary neoplasm breathlessness is often the most prominent symptom. Incoordination of the respiratory muscles of neurotic persons, especially when associated with palpitation, may be regarded as "a heart attack".

Conditions Causing Precordial Pain.

It is important to remember that cardiac pain is substernal in situation. The following conditions may cause pain which may be confused with that arising from heart disease.

Panniculitis.

Submammary pain on the left side is frequently noted in women, especially neurotic and highly strung women. Tenderness on pressure in the apical region is often present. These clinical phenomena arise in panniculitis (see Case XVIII).

Fibrositis.

When the intercostal muscles are affected, pain in the chest is noticed by the patient. This pain, which is aggravated by the increased respiratory effort caused by exertion and emotion and sometimes apparently by exposure to cold, may bear a superficial resemblance to *angina pectoris*. Sometimes the fibrositis may affect the

left *pectoralis major* muscle, and occasionally the pain is referred down the left arm. Fibrositic areas may be palpable and are usually tender on pressure and readily cleared up by deep massage (see Cases XIX to XXII). In some cases the condition of these patients improves after the administration of thyreoid substance or vitamin B_1.

Muscle Sprain.

Patients suffering from left pectoral muscle sprain are not infrequently encountered in workers' compensation practice. The history given by the patient is that he experienced a sharp pain in the chest just lateral to the left margin of the sternum after a severe muscular effort, such as that involved in lifting a heavy weight. As the exertion may cause palpitation and is often accompanied by breathlessness, the worker usually suspects that he has damaged his heart, and his suspicions unfortunately are often confirmed by his medical attendant, who diagnoses "heart strain". It is usually a matter of great difficulty to convince him that his heart is quite normal once the seeds of doubt have been implanted in his mind. In pectoral muscle sprain an area of tenderness to palpation is usually present in the region of the origin of the *pectoralis major* muscle. The pain from which the patient suffers is aggravated by the contraction of the affected muscles, and it may be some weeks before it finally disappears. Examination of the heart by the various methods outlined above shows it to be normal. Sometimes, however, the case is complicated by heart disease, and on occasion the muscles covering the posterior aspect of the chest are involved (see Cases XXIII and XXIV).

Pleurisy.

Diaphragmatic pleurisy sometimes produces pain which may be mistaken for cardiac pain; but the close relation of pleuritic pain to the phases of respiration should render the distinction easy (see Case XXV).

Pain of Gastric Distension.

Pain arising as a result of a distended stomach may suggest *angina pectoris*. The characteristic radiation is usually absent, and the pain is situated lower and is often aggravated by the taking of food. While cardiac pain sometimes occurs after a meal, the additional factors of exertion or emotion are usually necessary to produce it (see Cases XXVI and XXVII).

Other Conditions Causing Precordial Pain.

Among other conditions causing pain, *herpes zoster* may be mentioned. Sometimes the pain is severe before the occurrence of the vesicles, and often, especially in elderly subjects, a severe post-herpetic neuralgia persists long after the rash has disappeared. Referred pain arising from spondylitis of the thoracic portion of the spine should not be difficult to differentiate from cardiac pain; but difficulties may arise, because heart disease and spondylitis are frequently associated.

Conditions Causing Palpitation.

By palpitation is understood a consciousness of the heart's action. The beating of the heart usually intrudes upon consciousness as a result of some alteration of rhythm, rate or force. It may result from premature contractions, paroxysmal tachycardia, paroxysmal auricular flutter and paroxysmal auricular fibrillation. Among the extracardiac causes of palpitation may be mentioned tachycardia arising from emotional, febrile or digestive disturbances, tachycardia associated with thyreotoxicosis and tachycardia resulting from a cardiac neurosis or neuro-circulatory asthenia.

Neuro-Circulatory Asthenia.

Neuro-circulatory asthenia, variously known as Da Costa's syndrome, or "D.A.H." or the effort syndrome, has come into prominence again since the outbreak of the war, and numerous articles on the subject have appeared in the medical Press. The three Goulstonian lectures delivered in 1941 by Paul Wood are worthy of close study.

Certain aspects of the condition should be emphasized. It should be recognized that it is common in children and may occur in the elderly. It is frequently found in women (see Table II). Wood suggests that the failure to recognize this condition in civilian life may be due to the fact that it is commoner in women than in men. He states that "'effort syndrome' in the male soldier becomes cardiac, respiratory, or other neurosis in the female civilian". The most frequently noted symptoms of the condition are breathlessness, left thoracic pain, palpitation, fatigue, sweating and dizziness. The patients in the series studied exhibited these symptoms in various combinations (see Cases XXVIII to LX).

Breathlessness.—There appears to be no doubt that these patients experience a genuine sensation of breathlessness in circumstances which would not affect a normal person. The cause of the dyspnœa is uncertain; but it seems likely that the respiratory centre is affected by the emotional stimuli, and the dyspnœa is aggravated and prolonged by the phenomena resulting from hyperventilation (dizziness, paræsthesia, shakiness, mental confusion and tetany).

Left-Sided Thoracic Pain.—The pain in neuro-circulatory asthenia is situated in the left inframammary region, in contradistinction to the pain of cardiac disease, which is usually substernal in position. The mechanism by which this pain is produced is a matter of controversy. As it is associated with poor diaphragmatic movement and diminished thoracic expansion, it has been suggested that a functional disturbance of the respiratory muscles may be a factor in its production. The proponents of this theory, faced with the difficulty of explaining why the pain is predominantly left-sided, state that the pain, which is inaugurated by incessant minimal trauma from the over-acting nervous heart, is exaggerated by the fixed idea that the heart is at fault; the tendency is thus for the pain to be localized on the left side. Wood, after performing an interesting series of experiments, concluded that the left inframammary pain arose in local muscular or fibrous tissue.

Palpitation.—It should be remembered that tachycardia is a normal psychosomatic effect of emotion—that the physiological acceleration of the pulse rate after effort is not abnormal, and that there is no evidence that it is harmful. Wood emphasized that the pulse rate of normal controls during strenuous effort sometimes rose to 190 per minute without causing distress, and he stated that he had been informed by J. E. Lovelock, the famous mile runner, that his pulse rate sometimes reached 210 beats per minute. Wood also noted that persons suffering from Da Costa's syndrome were not hypersensitive to adrenaline, as judged by the pulse rate. He considered that this observation was of paramount importance, in that it showed that the heart did not overact to normal sympathetic stimulation, and that therefore it was not irritable. Palpitation in some cases, however, may be ascribed to premature contractions.

Other Symptoms.—The fatigue, the sweating (which is mostly confined to the palms of the hands, the soles of the feet and axillæ) and the dizziness are results of emotional stimuli. Physical examination as a rule reveals no serious abnormality; but the subjects affected by neuro-circulatory asthenia are usually nervous, and evidence of a feeble peripheral circulation is seen in their cold, blue extremities. Sweating from the palms and the axillæ is frequently noted. The systolic blood pressure is often raised, and tremors are frequently present. In some cases, however, the clinical features of the disorder are associated with organic disease of the heart.

Diagnosis.—With regard to the diagnosis of neuro-circulatory asthenia, it should be emphasized that this should be based on positive findings and should not be made by exclusion of other conditions. As Yellowlees[32] has stated, "if the old formula 'exclude organic disease before diagnosing neurosis' were amended to read: 'exclude neurosis (if you can) before diagnosing organic disease', it would go some way towards preventing the commonest error in medicine".

Causation.—The chief factors involved in the causation of neuro-circulatory asthenia are constitutional incapacity,

infection, neurosis, undue mental or physical strain and a family history with a high incidence of nervousness and neurosis.

Constitutional Incapacity: Wood found that an early history of nocturnal enuresis, phobias, tics, stammering and nightmares was common; but effort intolerance did not develop till the age of ten or eleven years. He found that the association of effort with the fear of being injured in games might initiate "a habit or conditioned response which maintains the symptoms when conscious fear is lost". It is of interest to note that Hurst,[17] while finding that the effort syndrome was particularly common among soldiers, noted that it was rare in the Air Force and particularly rare in the Navy.

Neurosis: Suggestion, either self-induced by the patient or resulting from a doctor's diagnosis of a damaged heart after some infection, may in susceptible subjects initiate the symptoms.

Treatment.—The treatment of the condition must be largely prophylactic. Wood makes the following statement:

Militia medical boards must learn to recognize personalities that will not stand up to military service: a few intelligent questions are worth ten effort-tolerance tests. The medical officers in the Services must learn to diagnose neurosis on positive grounds.

The essential trouble in the case of a person suffering from the symptoms of neuro-circulatory asthenia lies in his personality and mental make-up. Although the medical attendant cannot hope to alter materially such inborn characteristics, he can do much to help these people by not over-emphasizing minor defects of cardiac structure and function and by eliminating influences which perpetuate the conditioned responses to fear and effort, especially in the younger people. In the established case explanation of the symptoms as nervous manifestations, the use of sedatives, such as phenobarbital and bromide as required, and graduated exercise, are the best line of treatment, and it must be reinforced, as Conner[18] states, by "the liberal application of the two most generally effective remedies in the physician's armamentarium—reassurance and encouragement".

SUMMARY.

1. A description of the clinical criteria used in the diagnosis of the normal heart is given. Reference is made to some of the conditions simulating cardiac disease.

2. The effect of posture and of respiration in modifying auscultatory, radiological and electrocardiographic findings in the case of the normal heart is stressed.

3. The investigation of 15 normal medical students and of 45 patients suffering from conditions simulating cardiac disease is recorded.

ACKNOWLEDGEMENTS.

I wish to thank the 15 normal medical students for their willing cooperation in the investigation. I should also like to acknowledge my indebtedness to Dr. J. G. Edwards for his advice with regard to the X-ray examination of the heart, to Dr. T. J. Biggs for his assistance in obtaining some of the references, and to Mr. L. A. Hooke, general manager, Mr. Brooker and Mr. Giles, of Amalgamated Wireless (Australasia), Limited, for their cooperation in the production and demonstration of the record of the heart sounds.

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AN INVESTIGATION OF THE EFFECT OF ADMINISTRATION OF VITAMIN B_1 UPON GASTRIC SECRETION AND THE MOTOR ACTIVITIES OF THE STOMACH: A PRELIMINARY REPORT.

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Outline of the Investigation.

THIRTY-SIX patients attending the Royal Melbourne Hospital were selected; a test meal examination had revealed achlorhydria or hypochlorhydria in all of them. These patients then had the test meal examination repeated, but it was preceded by an injection of histamine to determine the degree of response to such a gastric stimulant. Each patient was subsequently given a barium meal, and the tone, peristalsis, degree of ptosis (if any) and emptying time of the stomach were determined by repeated X-ray screening observations. The examination being completed, the patient was given daily injections (intramuscularly) of 1,000 units of vitamin B_1 for ten consecutive days. In the case of the last seventeen patients the amount injected was 3,000 units per day. On the completion of these injections the patient was again given a fractional test meal examination and finally was subjected to another X-ray examination after a barium meal.

A comparison was thus instituted of the gastric secretion and motor activity of the stomach before and after this intensive course of vitamin B_1 therapy.

A group of seven medical students was used as a control series to determine the individual variation in emptying time of the stomach from day to day, and also the variation from student to student. A further group of four students was used to see what response, if any, was brought about by the administration of vitamin B_1 to young healthy subjects receiving an ordinary diet. This group of students underwent precisely the same routine examination as the

TABLE I.

Patient's Number and Age in Years.	Injection of Vitamin B_1.	Findings on Histamine Test Meal Examination.	Findings of Test Meals.		Findings on X-ray Examinations.	
			Before Vitamin B_1.	After Vitamin B_1.	Before Vitamin B_1.	After Vitamin B_1.
I 63	1,000 units each day for ten days.	8.5.41. HCl, nil. T.A., max. 5°.	7.4.41. HCl, nil. T.A., max. 8°. B., 1–1½ hours sp. incl., +. S., ½–1½ hours sp. incl., +. M., ½–1½ hours sp. incl., +.	20.4.41. T.A., max. 11°. B., nil. S., ½–1½ hours sp. incl., +. M., ½–1½ hours sp. incl., +.	9.4.41. T., orthohypotonic. P., normal. Pt., greater curvature 4 ins. below iliac crest; pylorus 1 in. below. E.T., 6 hours (merest trace left in stomach); head at hepatic flexure.	24.4.41. T., orthohypotonic. P., normal to active. Pt., greater curvature 3 ins. below iliac crest; pylorus about 1 in. below. E.T., 4 hours (just detectable trace in stomach); head in caecum.
II 64	1,000 units each day for ten days.	4.4.41. HCl, max. 25°. T.A., max. 25°.	2.4.41. HCl, max. 9°. T.A., max. 23°. B., ½–¾ hour sp. incl., +. S., ½–1½ hours sp. incl., +. M., ½–¾ hour sp. incl., +.	23.4.41. Free HCl, max. 10°. T.A., max. 30°. B., nil. S., ½–1½ hours sp. incl., +. M., ½–1½ hours sp. incl., +.	8.4.41. T., hypotonic. P., normal. Pt., greater curvature 5 ins. below iliac crest; pylorus 2 ins. below. E.T., 8½ hours (trace in stomach); head in caecum.	22.4.41. T., hypotonic. P., normal. Pt., greater curvature 5 ins. below iliac crest; pylorus 1·5 ins. below. E.T., 7 hours (trace in stomach); head in caecum.
III 59	1,000 units each day for ten days.	5.4.41. HCl, max. 25°. T.A., max. 45°.	4.4.41. HCl, nil. T.A., max. 25°. B., ½–1½ hours sp. incl., +. S., ½–1 hour sp. incl., +. M., nil.	26.4.41. HCl, nil. T.A., max. 25°. B., nil. S., ½–1 hour sp. incl., +. M., nil.	1.4.41. T., orthotonic. P., normal. Pt., greater curvature just below iliac crest; pylorus a little above. E.T., 4½ hours; head in middle part of ileum.	22.4.41. T., orthotonic. P., normal to active. Pt., greater curvature 2·5 ins. below iliac crest; pylorus at level of crest. E.T., 6 hours (merest trace in stomach); head in terminal part of ileum. *Note.*—Second barium meal examination put off for six days as patient was too ill.
IV 46	1,000 units each day for ten days.	21.2.41. HCl, max. 108°. T.A., max. 114°.	20.2.41. HCl, max. 9°. T.A., max. 34°. B., nil. S., ½–1½ hours sp. incl., +. M., ½ hour sp., +.	7.3.41. HCl, max. 20°. T.A., max. 48°. B., nil. S., ½–¾ hour sp. incl., +. M., nil.	25.2.41. T., orthotonic, P., normal. Pt., none. E.T., 4 hours.	5.3.41. T., orthotonic. P., normal. Pt., none. E.T., 3 hours.
V 60	1,000 units each day for ten days.	12.3.41. HCl, nil. T.A., max. 16°.	11.3.41. HCl, nil. T.A., max. 23°. B., nil. S., ½–1½ hours sp. incl., +. M., nil.	27.3.41. HCl, nil. T.A., max. 20°. B., nil. S., sp. 1–4 ++. M., nil.	13.3.41. T., orthotonic to hypotonic. P., normal. Pt., greater curvature 3·5 ins. below iliac crest; pylorus just above. E.T., 8½ hours (still a trace in stomach). Rather large stomach.	28.3.41. T., orthotonic. P., normal. Pt., greater curvature 2 ins. below iliac crest; pylorus just above. E.T., 5½ hours (merest trace in stomach). Normal sized stomach.
VI 45	1,000 units each day for ten days.	18.4.41. HCl, max. 24°. T.A., max. 38°.	7.4.41. HCl, nil. T.A., max. 12°. B., 1½–1½ hours sp. incl., +. S., ½–1½ hours sp. incl., +. M., nil.	1.5.41. T.A., max. 18°. B., ½–1½ hours sp. incl., +. S., ½–1½ hours sp. incl., +. M., nil.	8.4.41. T., orthotonic. P., normal. Pt., none; greater curvature at level of iliac crest. E.T., 2½–3 hours; head in caecum.	2.5.41. T., orthotonic. P., normal. Pt., none; greater curvature at level of iliac crest. E.T., 4½ hours (trace in stomach); head just in caecum.
VII 27	1,000 units each day for ten days.	29.4.41. HCl, 29°. T.A., 50°.	28.4.41. HCl, max. 10°. T.A., max. 46°. B., sp. 1–7 incl., +. S., sp. 1–4 incl., +. M., nil.	12.8.41. HCl, 26°. T.A., 54°. B., nil. S., sp. 1–6 incl., +. M., nil.	30.4.41. T., orthotonic. P., normal. Pt., greater curvature 2·5 or 3 ins. below iliac crest; pylorus a little above iliac crest. E.T., 7½ hours (merest trace); head in caecum; traces only 5 hours onward.	13.5.41. T., orthotonic. P., normal to active. Pt., greater curvature 4 ins. below iliac crest; pylorus just above iliac crest. E.T., 4 hours (merest trace); head in terminal part of ileum.
VIII 20 (Student)	1,000 units each day for ten days.	26.6.41. HCl, max. 45°. T.A., max. 63°.	25.6.41. HCl, max. 18°. T.A., max. 26°. B., sp. 4 and 5, +++. S., sp. 1–6 incl., +++. M., sp. 2–6 incl., +.	7.7.41. Free HCl, 28°. T.A., 42°. B., sp. 2–5 incl., +. S., sp. 1–6 incl., ++. M., nil.	27.6.41. T., orthotonic. P., active. Pt., greater curvature about 2 ins. below iliac crest; pylorus just above. E.T., 3½ hours (merest trace); head in hepatic flexure.	9.7.41. T., orthotonic. P., normal to active. Pt., greater curvature 2 ins. below iliac crest; pylorus at level of iliac crest. E.T., 3½ hours (merest trace); head in middle part of transverse colon.
IX 20 (Student)	1,000 units each day for ten days.	26.6.41. HCl, 59°. T.A., 84°.	25.6.41. HCl, max. 30°. T.A., max. 48°. B., nil. S., sp. 1–6 incl., +++. M., nil.	7.7.41. Free HCl, max. 43°. T.A., 70°. B., nil. S., sp. 1–6 incl., ++. M., nil.	27.6.41. T., orthotonic. P., normal. Pt., greater curvature 1 in. below iliac crest; pylorus 1·5 ins. above iliac crest. E.T., 5½ hours (stomach empty); head in caecum.	9.7.41. T., orthotonic. P., normal. Pt., greater curvature 3–4 ins. below iliac crest; pylorus at level of iliac crest. E.T., 4 hours (stomach empty); head in caecum.
X 20 (Student)	1,000 units each day for ten days.	16.7.41. HCl, max. 71°. T.A., max. 94°.	15.7.41. HCl, max. 44°. T.A., max. 66°. B., nil. S., sp. 1–5 incl. M., nil.	5.8.41. HCl, 44°. T.A., 68°. B., nil. S., sp. 1–6 incl., +++. M., nil.	21.7.41. T., orthotonic. P., active. Pt., none; greater curvature just above iliac crest. E.T., 4½ hours (stomach empty); head in splenic flexure.	7.8.41. T., orthotonic. P., normal to active. Pt., none; greater curvature just above iliac crest. E.T., 4½ hours (trace in stomach); head in hepatic flexure.
XI 20 (Student)	1,000 units each day for ten days.	16.7.41. HCl, max. 45°. (1st sp.). T.A., 66°.	15.7.41. HCl, max. 16°. T.A., max. 30°. B., sp. 1–3 incl., +. S., sp. 1–6 incl., +++. M., nil.	5.8.41. HCl, max. 18°. T.A., max. 34°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	22.7.41. T., high orthotonic. P., normal. Pt., none; greater curvature well above iliac crest. E.T., 3 hours (stomach empty); head in ileum.	7.8.41. T., hypertonic to orthotonic. P., normal. Pt., none; greater curvature well above iliac crest. E.T., 2½ hours (merest trace); head in ileum.
XII 61	1,000 units each day for ten days.	14.5.41. HCl, max. 9°. T.A., max. 30°.	30.4.41. HCl, nil. T.A., max. 32°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +++.	28.5.41. Free HCl, nil. T.A., max. 10°. B., sp. 3–5 incl., +. S., sp. 1–4 incl., +. M., sp. 2–6 incl., +.	17.5.41. T., orthotonic to hypotonic. P., normal to active. Pt., greater curvature 3·5–4 ins. below iliac crest; pylorus just below iliac crest; head in transverse portion of ileum. E.T., 2½ hours (merest trace).	31.5.41. T., orthotonic. P., normal to active. Pt., greater curvature 3–4 ins. below iliac crest; pylorus at level of iliac crest. E.T., 3½ hours (trace).

HCl = hydrochloric acid. T.A. = total acidity. max. = maximum. B. = bile. sp. incl. = specimens inclusive. Specimens collected at quarter-hour intervals. S. = starch. M. = mucus. T. = tone. P. = peristalsis. Pt. = ptosis. E.T. = emptying time.

TABLE I.—*Continued.*

Patient's Number and Age in Years.	Injection of Vitamin B_1.	Findings on Histamine Test Meal Examination.	Findings of Test Meals.		Findings on X-ray Examinations.	
			Before Vitamin B_1.	After Vitamin B_1.	Before Vitamin B_1.	After Vitamin B_1.
XIII 19	1,000 units each day for ten days.	31.7.41. HCl, max. 30°. T.A., max. 52°.	30.7.41. HCl, max. 18°. T.A., max. 27°. B., nil. S., sp. 1–5 incl., +. M., sp. 1–6 incl., +++.	15.8.41. HCl, max. 36°. T.A., max. 56°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	4.8.41. T., orthotonic. P., normal. Pt., greater curvature 1 in. below iliac crest; pylorus 1 in. above iliac crest. E.T., 5½ hours (stomach empty); head in caecum.	18.8.41. T., orthotonic. P., normal to active. Pt., greater curvature just to iliac crest. E.T., 3½ hours (stomach empty); head in terminal ileum.
XIV 60	1,000 units each day for ten days.	2.8.41. HCl, nil. T.A., max. 8°.	1.8.41. HCl, nil. T.A., max. 10°. B., sp. 4–5 incl., +. S., sp. 1–3 incl., ++. M., sp. 6, +.	22.8.41. HCl, nil. T.A., max. 18°. B., sp. 1, +. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	11.8.41. T., orthotonic (?) to hypotonic. P., normal to active. Pt., greater curvature 1 in. below iliac crest; pylorus at level of iliac crest. E.T., 2½ hours (merest trace); head in terminal ileum.	25.8.41. T., orthotonic. P., normal. Pt., greater curvature just below iliac crest; pylorus at level of iliac crest. E.T., 3 hours (stomach empty); head in terminal ileum.
XV 31	1,000 units each day for ten days.	12.8.41. HCl, max. 24°. T.A., max. 42°.	6.8.41. HCl, nil. T.A., 30°. B., nil. S., sp. 1–6 incl., ++. M., sp. 1–6 incl., ++.	23.8.41. HCl, nil. T.A., max. 13°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	13.8.41. T., orthotonic. P., normal to active. Pt., greater curvature well above iliac crest. E.T., 4½ hours (stomach empty) (trace at 3¹/₄ hours).	26.8.41. T., orthotonic. P., normal to active. Pt., greater curvature above iliac crest. E.T., 3¹/₄ hours (trace); head in ascending colon.
XVI 67	1,000 units each day for ten days.	2.8.41. HCl, nil. T.A., 14°.	1.8.41. HCl, nil. T.A., 12°. B., nil. S., sp. 1–4 incl., +. M., sp. 1–6 incl., ++.	23.8.41. HCl, nil. T.A., max. 22°. B., nil. S., sp. 1–4 incl., +. M., nil.	11.8.41. T., orthotonic. P., normal. Pt., greater curvature about 1 in. below the crest; pylorus at level of iliac crest. E.T., 4 hours (stomach empty).	25.8.41. T., orthotonic. P., normal to active. Pt., greater curvature 1·5 ins. below iliac crest; pylorus just above iliac crest. E.T., 3½ hours (stomach empty); head in terminal ileum.
XVII 59	1,000 units each day for ten days.	2.8.41. HCl, nil. T.A., 10°.	1.8.41. HCl, nil. T.A., 18°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–6 incl., ++.	23.8.41. HCl, nil. T.A., max. 12°. B., sp. 1–3 incl., +. S., sp. 1–3 incl., +. M., sp. 1–6 incl., +.	8.8.41. T., orthotonic. P., normal. Pt., greater curvature 1 in. below iliac crest; pylorus at level of iliac crest. E.T., 6½ hours (small residue), 8 hours (stomach empty); head in caecum.	T., orthotonic. P., normal. Pt., greater curvature 2 ins. below iliac crest; pylorus at level of iliac crest. E.T., 5½ hours (stomach empty); head in caecum.
XVIII 63	1,000 units each day for ten days.	13.8.41. HCl, nil. T.A., 12°.	12.8.41. HCl, nil. T.A., 12°. B., sp. 2–6 incl., +. S., sp. 1–6 incl., ++. M., nil.	28.8.41. Free HCl, nil. T.A., 10°. B., sp. 4, +. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	18.8.41. T., orthotonic (?) to hypotonic. P., active. Pt., greater curvature 1 in. below iliac crest; pylorus just above iliac crest. E.T., 3½ hours (stomach empty); head in caecum.	29.8.41. T., orthotonic. P., normal to active. Pt., greater curvature 1·5 ins. below iliac crest; pylorus at level of iliac crest. E.T., 2½ hours (merest trace); head in caecum.
XIX 57	1,000 units each day for ten days.	13.8.41. HCl, 12°. T.A., 32°.	12.8.41. HCl, nil. T.A., 12°. B., nil. S., sp. 1–6 incl., ++. M., nil.	28.8.41. Free HCl, nil. T.A., 13°. B., sp. 2–6 incl. S., sp. 1–4 incl., +. M., nil.	18.8.41. T., orthotonic to hypotonic. P., normal. Pt., greater curvature 3 ins. below iliac crest; pylorus at level of iliac crest. E.T., 3½ hours (merest trace); head in caecum.	29.8.41. T., orthotonic to hypotonic. P., normal. Pt., greater curvature 2·5 ins. below iliac crest; pylorus at level of iliac crest. E.T., 4¹/₄ hours (trace); head in caecum.
XX 74	1,000 units each day for ten days.	11.8.41. HCl, max. 30°. T.A., max. 48°.	6.8.41. HCl, nil. T.A., 20°. B., nil. S., sp. 1–5 incl., ++. M., sp. 1–3 incl., +.	1.9.41. Free HCl, nil. T.A., 22°. B., sp. 2–6 incl., +++. S., sp. 1–3 incl., +. M., nil.	13.8.41. T., orthotonic. P., normal to active. Pt., greater curvature 4 ins. below iliac crest; pylorus 1 in. below iliac crest. E.T., 3½ hours (stomach empty); head in terminal ileum; large gastric air-bubble.	29.8.41. T., orthotonic (?) to hypotonic. P., active. Pt., greater curvature 5 ins. below iliac crest; pylorus 1·5 ins. below. E.T., 2½ hours (merest trace); big gastric air-bubble.
XXI 67	1,000 units each day for ten days.	13.8.41. HCl, nil. T.A., 24°.	12.8.41. HCl, nil. T.A., max. 24°. B., sp. 1–6 incl., +. S., sp. 1–6 incl., +. M., nil.	3.9.41. HCl, nil. T.A., 11°. B., sp. 3–6 incl., +. S., sp. 1–6 incl., ++. M., sp. 1–6 incl., +.	18.8.41. T., orthotonic. P., normal to active. Pt., greater curvature 1 in. below iliac crest; pylorus a little above iliac crest. E.T., 4½ hours (least trace); head in caecum.	30.8.41. T., orthotonic. P., normal. Pt., greater curvature 1·5 ins. below iliac crest; pylorus 1 in. above iliac crest. E.T., 3½ hours (least trace); head just in caecum.
XXII 42	1,000 units each day for ten days.	29.8.41. HCl, 6°. T.A., 24°.	21.8.41. HCl, nil. T.A., 12°. B., sp. 1–6 incl., +. S., sp. 1–6 incl., +. M., nil.	29.9.41. Free HCl, nil. T.A., 11°. B., sp. 1, +. S., sp. 1–6 incl., +. M., nil.	26.8.41. T., orthotonic (?) to hypotonic. P., normal. Pt., greater curvature 2 ins. below iliac crest; pylorus 1 in. above. E.T., 3½ hours (stomach empty); head in terminal ileum.	30.9.41. T., orthotonic to hypotonic. P., normal. Pt., pylorus 1·5 ins. above iliac crest. E.T., 3½ hours (stomach empty); head in ileum.
XXIII 58	3,000 units each day for ten days.	10.9.41. HCl, 8°. T.A., 22°.	3.9.41. HCl, nil. T.A., 10°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–5 incl., +.	2.10.41. Free HCl, 18°. T.A., 30°. B., sp. 3, +. S., sp. 1–3 incl., ++. M., nil.	22.9.41. T., orthotonic. P., normal. Pt., greater curvature 0·5 to 1 in. below iliac crest. E.T., 2¹/₄ hours (trace); head in ileum.	3.10.41. T., orthotonic (?) to hypotonic. P., normal to active. Pt., greater curvature about 2 ins. below iliac crest; pylorus just above iliac crest. E.T., 1¹/₄ hours (stomach empty); head in terminal ileum.

HCl = hydrochloric acid. T.A. = total acidity. max. = maximum. B. = bile. sp. incl. = specimens inclusive. Specimens collected at quarter-hour intervals. S. = starch. M. = mucus. T. = tone. P. = peristalsis. Pt. = ptosis. E.T. = emptying time.

TABLE I.—*Continued.*

Patient's Number and Age in Years.	Injection of Vitamin B_1.	Findings on Histamine Test Meal Examination.	Findings of Test Meals.		Findings on X-ray Examinations.	
			Before Vitamin B_1.	After Vitamin B_1.	Before Vitamin B_1.	After Vitamin B_1.
XXIV 26	3,000 units each day for ten days.	10.9.41. HCl, 30°. T.A., 56°.	3.9.41. HCl, 7°. T.A., 22°. B., sp. 1–6 incl., +. S., sp. 1–6 incl., +. M., nil.	3.10.41. Free HCl, 5°. T.A., 23°. B., sp. 1–6 incl., +. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	23.9.41. T., orthotonic to hypotonic. P., normal. Pt., greater curvature 3 ins. below iliac crest; pylorus just above iliac crest. E.T., 6½ hours (trace); head in terminal ileum.	6.10.41. T., orthotonic (?) to hypotonic. P., normal to active. Pt., greater curvature 4 ins. below iliac crest; pylorus just below. E.T., 6 hours (merest trace); head in terminal ileum.
XXV 43	3,000 units each day for ten days.	4.9.41. HCl, 12°. T.A., 24°.	3.9.41. HCl, 10°. T.A., 25°. B., nil. S., sp. 1–6 incl., +. M., nil.	3.10.41. Free HCl, 24°. T.A., 39°. B., nil. S., sp. 1–4 incl., +. M., nil.	23.9.41. T., orthotonic. P., normal. Pt., greater curvature 1 in. below iliac crest; pylorus 1 in. above. E.T., 2½ hours (merest trace); head in ileum.	4.10.41. T., orthotonic. P., normal. Pt., greater curvature just to iliac crest. E.T., 2½ hours (tiny trace); head in terminal ileum.
XXVI 41	3,000 units each day for ten days.	10.9.41. HCl, 4°. T.A., 20°.	3.9.41. HCl, nil. T.A., 12°. B., sp. 1–6 incl., ++. S., sp. 1–6 incl., +. M., nil.	3.10.41. Free HCl, nil. T.A., 8°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	25.9.41. T., orthotonic to hypotonic. P., normal to active. Pt., greater curvature about 2 ins. below iliac crest; pylorus at level of iliac crest. E.T., 4 hours (merest trace); head in terminal ileum.	2.10.41. T., hypotonic. P., active. Pt., greater curvature 2·5 to 3 ins. below iliac crest; pylorus 0·5 in. below iliac crest. E.T., 4½ hours (merest trace); head in terminal ileum.
XXVII 45	3,000 units each day for ten days.	9.9.41. HCl, 17°. T.A., 38°.	7.9.41. HCl, nil. T.A., 22°. B., sp. 1–4 incl., +. S., sp. 1–6 incl., +. M., nil.	8.10.41. Free HCl, nil. T.A., 34°. B., sp. 1–6 incl., +. S., sp. 1–6 incl., +. M., nil.	24.9.41. T., orthotonic to hypotonic. P., normal to active. Pt., greater curvature 2 ins. below iliac crest; pylorus at level of iliac crest. E.T., 7½ hours (merest trace).	6.10.41. T., hypotonic. P., normal. Pt., greater curvature 4 ins. below iliac crest; pylorus 0·5 inch below iliac crest. E.T., 4½ hours (merest trace); head in terminal ileum.
XXVIII 58	1,000 units each day for ten days.		Only test meal examination was that following a course of vitamin B_1 treatment.	21.6.41. Free HCl, nil. T.A., 26°. B., nil. S., sp. 1–6 incl., +. M., sp. 2–6 incl., +.	27.8.41. T., hypotonic. P., normal to active. Pt., greater curvature 2·5 to 3 ins. below iliac crest; pylorus just above iliac crest. E.T., 3 hours (stomach empty); "preliminary meal" examination made 2 months after completion of injections.	24.6.41. T., hypotonic. P., normal. Pt., greater curvature about 3 ins. below iliac crest; pylorus about 1 in. above iliac crest. E.T., 4½ hours (stomach empty); head in caecum.
XXIX 19	3,000 units each day for ten days.	3.9.41. HCl, 10°. T.A., 26°.	27.8.41. HCl, nil. T.A., 13°. B., nil. S., sp. 1–6 incl., +. M., nil.	13.10.41. Free HCl, 32°. T.A., 47°. B., nil. S., sp. 1–6 incl., +. M., nil.	30.9.41. T., orthotonic. P., normal. Pt., greater curvature 1·5 ins. below iliac crest; pylorus 1·5 inches above iliac crest. E.T., 3½ hours (stomach empty); head in ileum.	14.10.41. T., orthotonic. P., active. Pt., greater curvature 1·5 ins. below iliac crest; pylorus 0·5 in. above iliac crest. E.T., 4 hours (trace); head in terminal ileum.
XXX 51	3,000 units each day for ten days.	18.9.41. HCl, 77°. T.A., 105°.	15.9.41. HCl, nil. T.A., 44°. B., sp. 1–6 incl., +. S., sp. 1–6 incl., +. M., sp. 1–4 incl., +.	6.10.41. Free HCl, 55°. T.A., 82°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	24.9.41. T., hypotonic. P., normal to active. Pt., greater curvature 3 ins. below iliac crest; pylorus at level of iliac crest. E.T., 3½ hours (trace); head in caecum.	4.10.41. T., hypotonic. P., normal. Pt., greater curvature 3·5 ins. below iliac crest; pylorus just below iliac crest. E.T., 4+ hours (less than 4½) (trace); head in terminal ileum.
XXXI 41	3,000 units each day for ten days.	11.9.41. HCl, 25°. T.A., 35°.	10.9.41. HCl, nil. T.A., 11°. B., sp. 6, +. S., sp. 1–6 incl., +. M., nil.	6.10.41. Free HCl, nil. T.A., 12°. B., sp. 1, +; sp. 3 and 4, +++. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	23.9.41. T., orthotonic. P., normal. Pt., none; greater curvature above iliac crest. E.T., 3½ hours (stomach empty); head in terminal ileum.	3.10.41. T., orthotonic. P., normal to active. Pt., none; greater curvature above iliac crest. E.T., 3½ hours (merest trace); head in terminal ileum.
XXXII 46	3,000 units each day for ten days.	23.9.41. HCl, 20°. T.A., 44°.	3.9.41. HCl, 15°. T.A., 28°. B., sp. 1, +. S., sp. 1–6 incl., +. M., sp. 1–3 incl., +.	6.10.41. Free HCl, 29°. T.A., 44°. B., sp. 1, +. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	23.9.41. T., orthotonic. P., normal to active. Pt., none; greater curvature just to iliac crest. E.T., 3½ hours (stomach empty); head in ileum.	4.10.41. T., hypotonic. P., normal. Pt., greater curvature 2·5 to 3 ins. below iliac crest; pylorus at level of iliac crest. E.T., 4 hours (stomach empty); head in terminal ileum.
XXXIII 49	3,000 units each day for ten days.	24.9.41. HCl, nil. T.A., 24°.	23.9.41. HCl, nil. T.A., 10°. B., nil. S., sp. 1–6 incl., ++. M., nil.	14.10.41. Free HCl, nil. T.A., 10°. B., nil. S., sp. 1–3 incl., +. M., sp. 1–6 incl., +++.	1.10.41. T., orthotonic (?) to hypotonic. P., normal. Pt., greater curvature 2 ins. below iliac crest; pylorus 1 in. above iliac crest. E.T., 2½ hours (merest trace); head in ileum.	13.10.41. T., orthotonic. P., normal to active. Pt., greater curvature 2 ins. below iliac crest; pylorus 0·5 in. above iliac crest. E.T., 3 hours (merest trace); head in caecum.
XXXIV 71	3,000 units each day for ten days.	24.9.41. HCl, 10°. T.A., 20°.	22.9.41. HCl, nil. T.A., 12°. B., nil. S., sp. 1–4 incl., +. M., sp. 1–4 incl., +.	16.10.41. HCl, nil. T.A., 12°. B., nil. S., sp. sp. 1–3 incl., +. M., nil.	26.9.41. T., orthotonic. P., active. Pt., greater curvature 1 in. below iliac crest; pylorus just above iliac crest. E.T., 3½ hours (trace in stomach); head in terminal ileum.	15.10.41. T., orthotonic. P., active. Pt., greater curvature 1 in. below iliac crest; pylorus 1 in. above iliac crest. E.T., 2½ hours (merest trace); head in terminal ileum.
XXXV 44	3,000 units each day for ten days.	25.9.41. HCl, 16°. T.A., 28°.	24.9.41. HCl, nil. T.A., 20°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	15.10.41. HCl, 12°. T.A., 25°. B., sp. 1–6 incl., +. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	3.10.41. T., orthotonic. P., normal to active. Pt., none; greater curvature above iliac crest. E.T., 2½ hours (merest trace); head in terminal ileum.	14.10.41. T., orthotonic. P., normal to active. Pt., none; greater curvature above iliac crest. E.T., 2½ hours (merest trace); head in caecum.

HCl = hydrochloric acid. T.A. = total acidity. max. = maximum. B. = bile. sp. incl. = specimens inclusive. Specimens collected at quarter-hour intervals. S. = starch. M. = mucus. T. = tone. P. = peristalsis. Pt. = ptosis. E.T. = emptying time.

TABLE I.—*Continued.*

Patient's Number and Age in Years.	Injection of Vitamin B_1.	Findings on Histamine Test Meal Examination.	Findings of Test Meals.		Findings on X-ray Examinations.	
			Before Vitamin B_1.	After Vitamin B_1.	Before Vitamin B_1.	After Vitamin B_1.
XXXVI 70	3,000 units each day for ten days.	7.10.41. HCl, 32°. T.A., 50°.	1.10.41. HCl, 18°. T.A., 32°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–6 incl., + + +.	29.10.41. HCl, nil. T.A., 33°. B., sp. 1–6 incl., +. S., sp. 1–6 incl., +. M., nil.	14.10.41. T., orthotonic. P., normal to active. Pt., greater curvature 1 in. below iliac crest; pylorus 0·5 in. above iliac crest. E.T., 2¾ hours (merest trace); head in terminal ileum.	28.10.41. T., orthotonic. P., normal. Pt., greater curvature 1 in. below iliac crest; pylorus 1 in. above iliac crest. E.T., 2½ hours (stomach empty); head in terminal ileum.
XXXVII 73	3,000 units each day for ten days.	6.10.41. HCl, 25°. T.A., 45°.	13.10.41. HCl, nil. T.A., 10°. B., sp. 2–6 incl., +. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	29.10.41. HCl, nil. T.A., 12°. B., nil. S., sp. 1–6 incl., +. M., sp. 1 and 2, +.	14.10.41. T., orthotonic. P., normal. Pt., greater curvature just to iliac crest. E.T., 2½ hours (merest trace); head in terminal ileum.	28.10.41. T., orthotonic. P., normal. Pt., greater curvature just to iliac crest. E.T., 2½ hours (stomach empty); head in terminal ileum.
XXXVIII 53	3,000 units each day for ten days.	15.10.41. HCl, 50°. T.A., 70°.	14.10.41. HCl, nil. T.A., 12°. B., sp. 1–6 incl., + +. S., sp. 1–3 incl., +. M., nil.	28.10.41. HCl, nil. T.A., 12°. B., sp. 1–5 incl., +. S., sp. 1–6 incl., +. M., nil.	17.10.41. T., orthotonic. P., normal to active. Pt., greater curvature 1 in. below iliac crest; pylorus 1 in. above iliac crest. E.T., 2½ hours (merest trace); head in ileum.	27.10.41. T., orthotonic. P., normal. Pt., greater curvature just above iliac crest. E.T., 2½ hours (merest trace); head in terminal ileum.
XXXIX 24	3,000 units each day for ten days.	7.10.41. HCl, 10°. T.A., 30°.	1.10.41. HCl, nil. T.A., 15°. B., sp. 3–6 incl., +. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	25.10.41. HCl, 11°. T.A., 28°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–6 incl., + + +.	13.10.41. T., orthotonic to hypotonic. P., normal to active. Pt., greater curvature 2 ins. below iliac crest; pylorus 0·5 in. above iliac crest. E.T., 4¼ hours (trace); head in ascending colon.	27.10.41. T., orthotonic. P., normal. Pt., greater curvature 3·5 ins. below iliac crest; pylorus at level of iliac crest. E.T., 4¼ hours (trace); head in caecum.
XL 60	3,000 units each day for ten days.	16.10.41. HCl, 40°. T.A., 50°.	13.10.41. HCl, nil. T.A., 20°. B., nil. S., sp. 1–6 incl., +. M., sp. 1–6 incl., +.	30.10.41. HCl, nil. T.A., 10°. B., sp. 5 and 6, +. S., sp. 1–4 incl., +. M., sp. 1–4 incl., +.	20.10.41. T., orthotonic. P., normal to active. Pt., greater curvature 1·5 ins. below iliac crest; pylorus 1 in. above iliac crest. E.T., 3 hours (merest trace); head in ileum.	31.10.41. T., orthotonic (?) to hypotonic. P., normal. Pt., greater curvature 1–2 ins. below iliac crest; pylorus 1 in. above iliac crest. E.T., 3½ hours (stomach empty), 3½ hours (trace in stomach); head in terminal ileum.

HCl = hydrochloric acid. T.A. = total acidity. max. = maximum. B. = bile. sp. incl. = specimens inclusive. Specimens collected at quarter-hour intervals. S. = starch. M. = mucus. T. = tone. P. = peristalsis Pt. = ptosis. E.T. = emptying time.

TABLE II.

Seven Student Controls: No Injections of Vitamin B_1. Barium Meal with X-ray Examination.

Name, Age and Date.	Tone.	Peristalsis.	Ptosis.	Emptying Time of Stomach.
H.H., 21 yrs.				
1.7.41 ..	Orthotonic.	Normal.	Greater curvature 1·5 inches below iliac crest; pylorus 1 inch above iliac crest.	4¼ hours (stomach empty); head in proximal part of transverse colon.
8.7.41 ..	Orthotonic (?) to hypotonic.	Normal (?) to active.	Greater curvature 2 to 3 inches below iliac crest; pylorus just above iliac crest.	4 hours (trace in stomach); head in caecum.
15.7.41 ..	Orthotonic (?) to hypotonic.	Normal.	Greater curvature 2·5 inches below iliac crest; pylorus 0·75 inch above iliac crest.	3¾ hours (merest trace); head in proximal part of transverse colon.
W.J., 20 yrs.				
26.6.41 ..	Orthotonic.	Normal.	Greater curvature at level of iliac crest.	4 hours (stomach empty); head in ileum.
3.7.41 ..	Orthotonic.	Normal.	Greater curvature just to iliac crest.	4½ hours (merest trace); head just in caecum.
10.7.41 ..	Orthotonic.	Normal.	Greater curvature just to iliac crest.	3¾ hours (stomach empty); head in terminal ileum.
S.Z., 20 yrs.				
26.6.41 ..	Orthotonic (?) to hypotonic.	Normal.	Greater curvature 2 inches below iliac crest; pylorus 1 inch above.	4½ hours (trace); head just in caecum.
3.7.41 ..	Hypotonic.	Normal.	Greater curvature 3·5 inches below iliac crest; pylorus at level of iliac crest.	5½ hours (small residue); head in ascending colon.
10.7.41 ..	Orthotonic (?) to hypotonic.	Normal to active.	Greater curvature 1 to 2 inches below iliac crest; pylorus 1 inch above iliac crest.	4½ hours (merest trace); head in caecum.
V., 20 yrs.				
26.6.41 ..	Orthotonic.	Normal.	Greater curvature at level of iliac crest.	4½ hours (merest trace); head in terminal ileum.
3.7.41 ..	Orthotonic.	Slow to normal.	Greater curvature at level of iliac crest.	3 hours (stomach empty); head in terminal ileum.
10.7.41 ..	Orthotonic.	Normal.	Greater curvature just above iliac crest.	3¼ hours (stomach empty); head in terminal ileum.
H.S., 20 yrs.				
1.7.41 ..	Orthotonic.	Normal.	Greater curvature just above iliac crest.	4¼ hours (stomach empty); head in proximal transverse colon.
10.7.41 ..	Orthotonic.	Active.	Greater curvature just above iliac crest.	4 hours (merest trace); head in caecum.
15.7.41 ..	Orthotonic.	Normal to active.	Greater curvature just to iliac crest.	3¾ hours (merest trace); head in caecum.
P.F., 21 yrs.				
1.7.41 ..	Orthotonic.	Normal to active.	Greater curvature just above iliac crest.	3½ hours (merest trace); head in caecum.
8.7.41 ..	Orthotonic.	Normal.	Greater curvature above iliac crest.	3½ hours (trace); head in ascending colon.
15.7.41 ..	Orthotonic.	Normal to active.	Greater curvature above iliac crest.	2¼ hours (merest trace); head in terminal ileum.
A.G., 20 yrs.				
1.7.41 ..	Orthotonic (?) to hypotonic.	Normal.	Greater curvature 1 inch below iliac crest; pylorus 0·5 inch above iliac crest.	3¼ hours (stomach empty); head just in caecum.
8.7.41 ..	Orthotonic.	Normal to active.	Greater curvature 2 inches below iliac crest; pylorus at level of iliac crest.	2¾ hours (least trace); head in terminal ileum.
15.7.41 ..	Orthotonic.	Normal.	Greater curvature 1 inch below iliac crest; pylorus just above iliac crest.	3¾ hours (merest trace); head in terminal ileum.

main group of Royal Melbourne Hospital patients, whereas the first group of students to which reference was made had barium meal X-ray examinations only and were considered entirely from the point of view of motor activity of the stomach.

The details of the findings in 40 subjects (four students and 36 patients) are given in Table I.

Discussion of Results.

Fractional Test Meals.

A careful consideration of the free hydrochloric acid and total acidity revealed no consistent change as the result of injections of vitamin B_1. The same conclusion applies to the presence of bile, starch and mucus in the gastric content.

Motor Activities of the Stomach.

No consistent or appreciable alteration of tone or of peristalsis was observed as the result of the administration of vitamin B_1, nor was the degree of ptosis, if present, modified. However, some interesting figures were obtained concerning the emptying time of the stomach as shown by X-ray examination. First let us consider the emptying time of the stomach of the seven healthy male students. The average figure obtained was 3 hours 56 minutes for the 21 barium meals. The greatest variation of emptying time for any student was 90 minutes and the smallest variation was 40 minutes. The average fluctuation for the seven students was 64 minutes, or 32 minutes on either side of an average emptying time of 3 hours 56 minutes. For practical purposes we may state that the average emptying time was four hours and that the approximate range of fluctuation was one hour.

In the consideration of the 36 hospital patients and four students who were subjected to detailed investigation, nine showed a decrease of gastric emptying time of more than one hour, whereas four showed an increase in gastric emptying time greater than one hour.

Details are seen in Tables III and IV.

TABLE III.

Patients showing Diminished Emptying Time of the Stomach after Administration of Vitamin B_1.

Number of Patient.	Emptying Time before Treatment with Vitamin B_1. (Hours.)	Emptying Time after Treatment with Vitamin B_1. (Hours.)	Diminution in Emptying Time. (Hours.)
1	6	4	2
2	8½	7	1½
5	8½	5½	2½
7	7½	4	3½
9	5½	4	1½
13	5½	3½	1½
17	6½	5	1½
18	3½	2	1½
27	7½	4½	3

TABLE IV.

Patients showing Increased Emptying Time of the Stomach after Administration of Vitamin B_1.

Number of Patient.	Emptying Time before Treatment with Vitamin B_1. (Hours.)	Emptying Time after Treatment with Vitamin B_1. (Hours.)	Increase in Emptying Time. (Hours.)
8	4½	6	1½[1]
6	2½	4½	1½
10	3½	4½	1½
28	3	4½	1½

[1] Owing to indisposition of the patient, the administration of a barium meal was delayed for six days after completion of the injections of vitamin B_1.

At first sight it would appear that such a small number (nine), in a total of 40 subjects, showing decrease in emptying time of the stomach has little significance, particularly as several others (four) actually showed an increase in emptying time after the administration of vitamin B_1. On closer inspection of the figures, however, it is revealed that with one exception (number 18) the emptying time of the stomach was very long—namely, 6, 8½, 7½, 5½, 5½, 6½ and 7½ hours. In such circumstances, apparently, administration of vitamin B_1 had considerable influence on increasing the rate at which the stomach was emptied.

Turning to the figures for the four patients whose gastric emptying time was actually greater after vitamin B_1 administration, we find that with one exception (number 3, who owing to indisposition did not have his barium meal until six days after the vitamin B_1 injections and therefore at a time when their influence may have waned somewhat) the emptying time was rapid—namely, 2½, 3½ and 3 hours—and the final emptying times (4½, 4½ and 4 hours), although greater than before treatment, were not very much greater than the normal emptying time of the stomach (4 hours).

Thus it seems that vitamin B_1 may have a distinct effect in hastening the emptying time of the stomach of those persons in whom it is habitually much delayed.

Of the 27 persons whose gastric emptying time was not affected to the extent of one hour by the administration of vitamin B_1, it will be observed that only one (number 24, with an emptying time of [illegible] hours) showed in the preliminary barium meal examination any great variation from normal.

Conclusions.[1]

1. The administration of vitamin B_1 appears to have no appreciable effect on gastric secretion in man.
2. Vitamin B_1 hastens the emptying time of the stomach of those persons whose gastric emptying time is habitually much longer than normal, but does not influence the rate of evacuation of the stomach of those whose gastric emptying time is normal or excessively rapid.

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We should also record our appreciation of the generosity of Parke, Davis and Company, who supplied much of the vitamin B_1 as "Vibex".

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[1] Further work on this subject is indicated. A team of investigators, including radiologist, biochemist, dietitian and physician, had been appointed for this purpose when the grave nature of international affairs caused the research to be deferred until after the war.

SCHOOL CHILDREN'S TEETH.[1]

By MARJORIE CASLEY SMITH,
Medical Inspector of Schools in the Education Department of South Australia.

I HAVE been asked to give a quick survey of our efforts in the Education Department on behalf of the school children's teeth, and of some of our findings in regard to them.

The medical staff of the department inspects both city and country schools. It is the present aim for every child to have a medical examination in the first year at school and then again when he reaches the fourth and seventh grades. Two further examinations are made at the high schools. The condition of the teeth is, of course, included in these. When dental caries is found, a notice is sent asking the parent to take the child to a dentist. Orders for the Dental Hospital are also sent to the children of the unemployed. There the matter rests, so far as we are concerned, until the next inspection of that child in three years' time. Practically, therefore, the whole responsibility for the care of the teeth lies with the parents, and depends largely on their financial position. The country schools are also visited periodically by a medical officer, a whole school being inspected at once. As there are many schools and only one officer, the visits are necessarily few and far between.

Apart from the medical officers, the Education Department has made it a part of its programme to have school dentists who visit the schools, particularly the country ones. Until 1927 a clinic was also conducted in the city; but it was then decided to increase the staff of the Dental Hospital so that that institution would do the work formerly done by the clinic. However, for some years past the hospital has been so crowded that only the unemployed have been treated there; but at present the families of men serving in the Army and Air Force are also accepted. Until the middle of this year, two women dentists were attached to the staff of the Education Department. Unfortunately we have now lost them both, and up to date have been able to replace only one of them.

It is customary for the school dentists to visit a country district and treat, free of cost, all the children there who cannot afford to pay for dental treatment, if the parents will consent. This is a long job, and therefore the districts can be visited only about once in three years—a most unsatisfactory arrangement for both the dentists and the patients, as figures will show.

In 1939 over 3,000 examinations were made and over 1,400 children were treated.

In 1940 the dentists examined 3,838 children; 977 were actually treated by them. This number does not include the children whose parents could afford other means of obtaining the fillings *et cetera* that were necessary, or who refused to have anything done. These figures are from the Mount Gambier and Streaky Bay districts alone, the examinations having taken one year to complete. Of these treatments in 1940, 1,897 were fillings of permanent teeth, and 527 were extractions of permanent teeth. Fillings of deciduous teeth numbered 578 and extractions 2,201.

In Adelaide and the suburbs, in round numbers 11,000 children were medically examined, with the result that 7,000 dental notices were sent out in 1940. In the previous year, out of 8,500 children seen, 5,250 had dental caries. Therefore in the last two years an average of 63% of the children examined had dental trouble—and in most cases bad trouble. I can state from my own experience that it is unusual to find only one tooth needing attention in children aged twelve years and over. Many, by that age, have cavities in their teeth, especially in the six year old molar if they still have it, which makes extraction the only possible course to adopt. Frequently, by the time high school is reached, upper incisors are badly decayed or absent, and double sets of dentures have already made their appearance in several of the mouths inspected each year. In these schools alone in the last two years, from 35% to 38% of the children examined had dental notices sent them.

A report by Dr. Sandford Morgan on the teeth of the children at the free kindergartens stated that one child in three was in need of attention. This bears out our figures as we see them in our schools a year or so later, when the condition is further advanced.

The causes? Ignorance and economic disability.

For my own interest, I made a graph of my figures of pale, malnourished children and of those with dental decay found at various schools. The numbers are too small to be of real value; but there is a definite rise in both malnutrition and decay in our poorer schools. As is to be expected, there is a rise of 10% in malnutrition and of 10% to 20% in dental caries. The high schools also, with two exceptions, show this correlation as well.

Medically speaking, the whole problem of decaying teeth and dirty, infected mouths is urgent and important. When we read an article such as that by K. Malik, L.D.S., M.R.C.S. (England), L.R.C.P. (London), in the *Medical World*, in which he states that it has been proved that in 6% of cases the saliva over the stumps of dead teeth contains hæmolytic streptococci, and that they are found in 30% of the apices of these stumps, then the reason for much of the gastric trouble and rheumatism in young people may well be in their teeth, rather than in their falsely suspected tonsils.

And what else is the Department doing in trying to improve these conditions? For the most part its work is along educational lines. Teachers are taught dental hygiene and the principles of diet. They are instructed to hold toothbrush drills and to give lessons on the care of teeth. Talks are given to mothers' clubs on the same lines. This year we have utilized the radio for a series of health talks. It is found necessary to instruct parents on the need for care of the first dentition, which is generally neglected, and also to awaken them to the need for the correction of irregularities in the permanent teeth.

But, as I have mentioned before, the lack of money to take the child to the dentist is a very real problem in many poor homes. A family of four, with the father receiving the basic wage, has very little money for the luxuries of the dental chair. And what amenities are offered to people in this class—the basic wage earner, the poorly paid clerk, the washer-woman? There are the registered dentists, who have their charges of 5s. 6d. or 10s. 6d. as the case may be. There is the Dental Hospital on Frome Road. Here, as has been previously stated, until the war the number of applicants was so great for the facilities available that only the unemployed could be taken. Now, with higher wages, things are easier there, and families of men in the Army and Air Force are also treated.

However, it is obvious that, in normal times, nothing remains for the poor family but neglect. Of course, if the tooth is aching too badly, and Tommy makes an awful fuss about it, there are a few people who will pull it out for a shilling. The expense of a filling is simply not to be thought of.

The problem of the outback also looms large upon the horizon. Shall we eventually have flying dentists, with their drills attached to the battery of the aeroplane?

In New Zealand an attempt to solve the problem of overcrowding at a central hospital has been made by establishing dental clinics dealing with children going to the schools in a given district; 324 of these have been established. They are controlled by a Dental Committee formed by local residents, who raise some of the money necessary to run them. School dental hygienists give the treatment necessary.

The suggestion made by the National Health and Medical Research Council regarding the institution of small public hospitals to be situated in the suburbs might possibly be enlarged to include dental services also; this would relieve congestion, the patients paying some small fee for treatment if they could.

A friendly society system along medical lines might also be formed to assist the lower wage earner to obtain proper dental care for his family.

[1] Read at a meeting of the Medical Sciences Club of South Australia in November, 1941.

Reports of Cases.

A CASE OF APPENDICEAL ABSCESS WITH UNUSUAL SYMPTOMS.

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Clinical Record.

L.C., a female child, aged one year and three months, on May 17, 1942, had an attack of vomiting and was fretful. During the examination the child was crying most of the time and it was impossible to elicit the presence of any tenderness or rigidity. The temperature was 98° F. and further examination failed to reveal any abnormality. The child was admitted to hospital for observation, and after three days, in the absence of any further developments, she was discharged.

On May 24 the child was brought to my rooms, the mother demonstrating a blood-stained napkin. The child was not crying or distressed in any way; she had not vomited since her discharge from hospital. On examination, no rigidity or tenderness was present and no mass was palpable in the abdomen. The temperature was 99·4° F., the pulse rate was 120 per minute, and the respirations numbered 24 per minute. The child was again placed under observation and during the night passed two blood-stained, loose motions. On examination the following morning she was seen to be lying in four ounces of almost pure dark clotted blood. At this time the temperature was 97° F., the pulse rate 130 per minute, and the respirations numbered 28 per minute. Examination still failed to reveal any tenderness or mass in the abdomen, and it was considered that the child possibly had an intussusception. However, no empty feeling could be elicited in the right iliac fossa. A rectal examination failed to reveal any mass. An attempt to give the child a barium enema failed, but she returned the fluid as fast as it was administered. By this time the general condition was still fair, but the child was beginning to look exsanguinated. It was decided that a blood transfusion should be given. The father's blood was found to be compatible, and 200 cubic centimetres were collected. The child was taken to the operating theatre, after having been given premedication of one-eightieth of a grain of morphine, and under nitrous oxide, oxygen and ether anæsthesia a laparotomy was performed. Two fingers were inserted through a small mid-line incision, and no mass was palpable; but as the fingers were exploring the right iliac fossa thick pus welled up. It was then recognized that the lesion was an appendiceal abscess, and a tube was placed in the right iliac fossa through the stab wound. Sulphanilamide powder was dusted into the peritoneal cavity. The wounds were closed with catgut and silkworm sutures, after which the patient was returned to the ward in a poor condition.

Meanwhile attempts had been made to give the blood transfusion, but they were unsuccessful in the absence of veins, even though four attempts were made to cut down on the recognized regular veins. Subsequently the blood was given intramuscularly into the buttocks, for its iron value.

The subsequent history is interesting in view of the light which it throws on the case. In order to give supplementary fluid, which was taken sparingly by mouth, glucose and saline solution were given continuously by rectum throughout the next twenty-four hours. Oxygen was also administered continuously for the same period. Morphine (one-eightieth of a grain) was given at intervals of approximately four to six hours. Sulphapyridine (one-third of an ampoule) was given intramuscularly at intervals of six hours for three days. One tablet of sulphapyridine was given at intervals of six hours until May 31. On May 27, as very little discharge was coming through the tube, it was removed. For two days a greyish discharge in small quantities continued to ooze from the stab wound. On the same day blood was again discharged from the rectum. This passage of blood *per rectum* continued at intervals for seven days; sometimes the blood was mixed with fæces, but it was always dark in colour. Atropine was given hypodermically (one two-hundredth of a grain every four hours); but the only measure that was found effective to control the hæmorrhage was barium solution as used for opaque enemata. Iron was administered in the form of *Syrupus Ferri Phosphatis Compositus*. The hæmoglobin value of the child's blood was 21%.

On June 2 there was some blood-stained discharge from the stab wound. This blood, however, was bright in colour, in contradistinction to the dark blood coming from the rectum. This was succeeded by fæcal discharge through the stab wound, which persisted until June 28. During the early stages of the fæcal discharge some constipation was present, but eventually the bowels opened normally and the stab wound then healed. The child was discharged from hospital on July 1, the mid-line incision having healed by second intention.

Discussion.

The interesting feature of this case is the unusual symptom of severe and repeated melena and hæmorrhage from the wound after the establishment of the temporary fæcal fistula; this indicates that a large vessel must have ruptured from the submucosa into the lumen of the cæcum, thus causing the melena. When drainage was established the blood passed from the cæcum to the abscess cavity, and so to the exterior through the stab wound.

Reviews.

DISEASES OF THE EYE.

ON the bookshelf the tenth edition (1942) of Sir John Parsons's "Diseases of the Eye" will look very much like its predecessors.[1] The binding and general get up of the book follow those of all preceding editions, save that, as might be expected, and, as is becoming, there is a tendency to a middle-age spread (first edition, 1907, aged thirty-five years).

In the preface Sir John Parsons speaks of the character of the book as being a reliable introduction to ophthalmology for students, general practitioners and junior ophthalmic surgeons. Actually the book is rather more than that, as it is quite authoritative upon certain matters and many a time has been found useful by ophthalmic surgeons as a work of reference. We venture to think that the book now tends to fall between two stools, in being rather too comprehensive for undergraduates and yet not complete enough as a reference work, and we do not consider that Mr. H. B. Stallard, whose name now appears on the title page, would have great difficulty in transforming future editions into just such a mine of wealthy information and tit-bits of clinical knowledge and findings as certain older books used to be in their days of glory—truly a satisfying work of reference.

In this edition there is no such grouping as tropical ophthalmology; this is a shortcoming. Everyone knows there is a good deal of misnomer about that term, yet is sapient enough to know that it implies references to those diseases which are frequent in the hot climates and others which are rare except in the more temperate climates. The book will be carried to all parts of the globe; there is no better book for an ophthalmologist to carry in limited baggage and such a chapter could be very helpful.

It would ill become the faithful old worshippers at the shrine of "Parsons" to look for the mote, yet mention may be made of a weird phrase on page 622, line 7, in regard to recurrent hordeola: "Inquiry should be made as to the condition of the drains." Which? Lachrymal, Meibomian, or sewerage? If the reference is to the last-mentioned, this surely is a mediæval tradition.

Australian surgeons do not handle pterygium by destroying the apex with diathermy, nor keep the McReynolds operation for recurrences. Keratoplasty is dealt with in some five lines, and corneal ulcers seem to have treatment with atropine and lotions rather than with additional drops of the silver or mercurial preparations.

There is a sorry lack of information upon the microscopy of the living eye and of the technique of the slit lamp. As the bricklayer needs his trowel, so does the modern ophthalmologist need his slit lamp—he is inefficient and prehistoric without one and without the minutiæ of detail and information the intelligent use of the apparatus can and should give him.

So, great and splendid as is this tenth edition of a distinguished and popular manual, and noble as is its excellent portrayal of the bookmaker's art in this third year of the war and all that that has meant to London and the printing house of J. & A. Churchill Limited, Portman Square, yet withal may we ask that the next edition be still better, even though it must then become larger.

[1] "Diseases of the Eye", by Sir John Herbert Parsons, C.B.E., F.R.C.S., F.R.S.; Tenth Edition; 1942. London: J. & A. Churchill Limited. Medium 8vo, pp. 754 with 21 plates and 372 text figures. Price: 25s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 19, 1942.

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ANÆMIA IN PREGNANCY: AN INDIAN STUDY.

PREGNANCY is a normal physiological condition. The pregnant woman passes through a time of stress; in addition to supplying her own needs, she has to meet the requirements of the developing fœtus. If she is in good health and orders her life with care in the matter of diet, exercise and so on, the end of the puerperium finds her none the worse for her life-giving achievement. Certain changes take place in the body of a pregnant woman and these are necessarily shared by the blood, since the blood stream is the medium of communication between mother and fœtus. A valuable discussion on the blood changes of normal pregnant women will be found in a series of articles by W. J. Dieckmann and C. R. Wegner[1] published in 1934. These authors who reviewed the literature and reported investigations of their own, showed, as others have also shown, that in pregnancy there is an increase in plasma volume and also an increase in the total number of red cells and in the hæmoglobin. The increase in plasma volume is greater than the increase in erythrocytes and hæmoglobin and this means that there is a relative decrease in erythrocytes and hæmoglobin. It is estimated that the plasma volume is increased during pregnancy by 25% or more, the total hæmoglobin by 13% and the total corpuscular volume by 20% or more. Thus it is apparent that what is known as the physiological anæmia of pregnancy is due to a condition of hydræmia. L. E. H. Whitby and C. J. C. Britton[2] refer to the suggestion that the hydræmia is part of the mechanism by which gaseous exchange between mother and fœtus is carried out with the minimal expenditure of work. They add that the same conditions allow of a relatively large loss of blood during labour, for these conditions tend to conserve hæmoglobin, whilst the large volume of fluid present in the tissues during pregnancy is rapidly absorbed after delivery and so restores the blood volume. Whitby and Britton conclude that figures for hæmoglobin as low as 10 grammes *per centum* (70% Haldane) and for erythrocytes 4,000,000 per cubic millimetre constitute the physiological anæmia of pregnancy and should not be regarded as pathological. A reduction in hæmoglobin below 10 grammes *per centum* (70% Haldane) constitutes an anæmia.

Anæmia in pregnancy, not the so-called physiological anæmia, is a subject which should receive attention at the present time when there is a shortage of certain articles of diet. F. H. Bethel in 1936 reported a study[3] of 66 healthy young women whom he observed during the last trimester of pregnancy; he used as controls 50 healthy non-pregnant women of the same age group. The blood values of 70% of the pregnant women (Bethel expressed his results in percentages) were too low to be accounted for solely by "physiological anæmia". He concluded that anæmia in pregnancy is commonly due either to preexisting iron depletion or to an inadequate intake of suitable protein during gestation. The report, therefore, of a recent investigation which comes from the School of Tropical Medicine, Calcutta, should not be allowed to pass unnoticed.[4] This report, which is by L. E. Napier and M. I. Neal Edwards, covers 135 pages and contains many graphs and tables. In addition to the 529 pregnant women, Napier and Edwards examined 128 "normal" non-pregnant women. It was found that little change in the blood findings were noted when the non-pregnant subjects passed the age of fifteen years. Of the 529 pregnant women 64 were regarded as normal and 465 as anæmic. When the 465 women were classified according to the degree of their anæmia it was found that the number of controls (the 64 "normal" pregnant women were controls) could be increased. The 465 anæmic pregnant women were divided into three classes. Class A, described as markedly anæmic, had a hæmoglobin percentage of below 50; Class B, described as moderately anæmic, had percentage figures above 50 but below 68; Class C, described as controls, had a hæmoglobin percentage of 68 and over. One-third of the women fell into this "control group". They gave the same range of hæmoglobin readings as those pregnant women placed in the normal group, but none of them could be considered normal from the clinical point of view; they felt ill, looked ill and appeared to be anæmic. The cases were subdivided also into microcytic, normocytic and macrocytic. When it is realized that epidemiological, pathological and clinical data were analysed with regard to the nine groups, some idea of the wealth of detail presented by this report may be obtained. The examination of this detail is the province of hæmatologists and cannot be attempted in this place; some of the more important observations only can be mentioned. There was apparent a tendency towards increase in the size of the erythrocyte as pregnancy advanced; at the same time macrocytic and microcytic anæmias usually maintained their own characteristics until cure was effected. An interesting observation was made in regard to some of the women in Class C, that is, those whose hæmoglobin

[1] W. J. Dieckmann and C. R. Wegner: *Archives of Internal Medicine*, Volume LIII, 1934, pages 71, 188, 345, 353, 527 and 540.

[2] L. E. H. Whitby and C. J. C. Britton: "Disorders of the Blood", Third Edition, 1939, page 247.

[3] F. H. Bethel: "The Blood Changes in Normal Pregnancy and Their Relation to the Iron and Protein Supplied by the Diet", *The Journal of the American Medical Association*, Volume CVII, 1936, page 564.

[4] L. E. Napier and M. I. Neal Edwards: "Anæmia in Pregnancy in Calcutta: An Analysis of Hæmatological and Other Data from 529 Pregnant Women", "Indian Medical Research Memoirs", Supplementary Series to the *Indian Journal of Medical Research*, Memoir Number 33, December, 1941.

percentage was 68 or above that level. In 95 cases later examinations showed that 82 remained in the same hæmoglobin class, four fell into the next class before parturition and nine fell *post partum* into the next class. This is the group which was regarded as being practically identical with the normal pregnancy group. The observation is made that had the 64 "normal" women been followed, some of them might have been found with hæmoglobin percentages of the order of Class B. Apart from the tendency of the cells to increase in size as pregnancy advanced, and also to move towards the normocytic and orthochromic group as improvement in the hæmoglobin percentage occurred, there was no evidence of the cyclical changes reported by some authors. Napier and Edwards found evidence that the removal of the fœtus, naturally at term or prematurely, was a more potent factor than any treatment that might have been given in bringing about an improvement in the blood picture; this was found particularly in the macrocytic and normocytic cases, but much less in those falling into the microcytic group. This observation makes one turn to the question of iron deficiency and its relationship to microcytic and macrocytic forms of anæmia as observed by Napier and Edwards. In the whole series they found evidence of iron deficiency. After weighing the evidence they conclude that the iron intake is insufficient for the extra requirements of the pregnant woman. The fact that there was a higher incidence of anæmia in the lower economic groups supports this view. Napier and Edwards observe that the fact that this "poverty" anæmia is not predominantly microcytic is not necessarily against the view, for the good reason that in all probability "the microcytic picture of the anæmia is overshadowed by other influences". A relative iron deficiency occurring in the child-bearing period of life with a tendency to exacerbations during pregnancy and lactation is recognized in other countries than India and is especially common among the poor whose iron intake is low. In Napier and Edwards's series the pure microcytic anæmia occurred mainly in *multiparæ* and the older age groups, suggesting to them that possibly iron depletion during an earlier pregnancy was the cause. Microcytic anæmia in their series appeared to be associated with the observance of *purdah*, the curious Indian custom of screening women from the sight of strangers. The pallor and debility which result from close confinement to the house and lack of fresh air and sunlight probably produce a condition similar to the chlorosis of a previous generation which responded quickly to iron therapy. In spite of all this, Napier and Edwards point out that iron deficiency, though widespread, is of secondary importance in their series of cases of anæmia, for the majority of cases and the most severe cases were macrocytic. What then is the cause of the macrocytic form of anæmia described by Napier and Edwards? At this stage it may be pointed out that Whitby and Britton divide the macrocytic anæmias of pregnancy into two groups, macrocytic anæmia complicated by pregnancy and macrocytic anæmia induced by pregnancy. The first of these they subdivide into tropical nutritional anæmia and true pernicious anæmia complicated by pregnancy. Napier and Edwards make no such classification. Rather in their discussion of macrocytic anæmia of this type do they cover most of the ætiological considerations to which attention would be drawn in Whitby and Britton's two main subdivisions. In the first place the production of macrocytic anæmia is, they hold, connected in some way with the presence of the fœtus—"the anæmia and the macrocytic tendency show a progressive increase throughout pregnancy, both in the anæmic patients and in the controls, and they tend to decrease immediately after parturition". The second factor causing macrocytic anæmia is in Napier and Edwards's opinion nutritional in origin; this is suggested not only by the higher incidence in the lower economic classes, but by the fact that it is the macrocytic type which is more closely correlated with low economic status, and that in the richer classes vegetarianism, with its lower protein intake, is correlated positively with macrocytic anæmia. The common association of macrocytic anæmia with sore mouth (a deficiency condition) still further suggests a nutritional defect. "Thirdly, the association of severe macrocytic anæmia with primigravidity, independent of age, its occurrence relatively early in pregnancy, and its association with premature delivery and a high neonatal death rate, suggest comparison with the toxæmias of pregnancy." In one point Napier and Edwards appear to be at direct variance with Whitby and Britton, and the reader wishes that they had subdivided macrocytic anæmia of pregnancy into groups as Whitby and Britton have done. Napier and Edwards state that the significant positive correlation of severe macrocytic anæmia with enlarged spleen and liver, hyperbilirubinæmia and reticulocytosis all point to some hæmolytic condition and are specially suggestive of chronic malaria. By this they do not mean that recently acquired malaria is the cause, but chronic or "latent" malaria with hyperreticuloendotheliosis. Their definition may be quoted *verbatim*:

> Findings seem to suggest that the macrocytic anæmia of pregnancy is a 'conditioned' toxæmia, that is, a toxæmia associated with the presence of the fœtus and conditioned by a low dietary intake, or deficient absorption, of certain essential blood-forming and protecting substances, the syndrome being aggravated by (or perhaps only operative in the presence of) a chronic malarial infection, with the associated hyper-reticulo-endotheliosis and excessive blood destruction which causes an extra demand for these blood-forming essentials, by chronic intestinal infections and diarrhœa with consequent exaggeration of mal-absorption, and/or by syphilis with its hæmopoietic depressing action.

Whitby and Britton, in discussing macrocytic anæmia induced by pregnancy, state that the older theories which attributed the macrocytic anæmia of pregnancy to a hæmolytic or toxic agent have been discarded. They point out that hæmolysis is not a feature of the disease and express the opinion that it would be advisable to discard the term "hæmolytic anæmia of pregnancy". In regard to tropical nutritional anæmia, Whitby and Britton state that the essential cause of the anæmia complicating pregnancy in India is the dietetic deficiency of the average native Indian and the subsidiary factors of malaria and hookworm infections. Malarial infection is, of course, one of the causes of hæmolytic anæmia and it is to be noted that in quite a number of their cases of severe anæmia (Class A) Napier and Edwards obtained an indirect reaction to the Van den Bergh test, a reaction obtained in hæmolytic anæmia. We may therefore conclude that Napier and Edwards are not at variance with Whitby and Britton. In spite of the fact that they give the mere presence of the fœtus as one of the causes of their cases of macrocytic anæmia, most of these cases probably belong to the tropical nutritional anæmia group and not to that of macrocytic anæmia induced by pregnancy.

From the account that has been given of the work of Napier and Edwards the importance of nutrition in pregnancy is obvious. The remedy for anæmia of pregnancy lies largely in its prevention. There should be little difficulty in Australia in avoiding nutritional anæmia of pregnancy, but practitioners, particularly in tropical parts of the Commonwealth, will be well advised to be watchful and prepared.

Current Comment.

SENSITIVITY TO CATGUT.

THE fact that a sensitivity to catgut may develop when relatively large amounts of catgut are used has recently been discussed by H. T. Langston[1] and the old argument of absorbable *versus* non-absorbable ligature and suture material has been revived. Such sensitivity may be readily demonstrated by patch or intradermal tests and it is suggested that in sensitized persons lysis of the suture material occurs more rapidly than in the non-sensitized. Strangely enough, sensitization to catgut appears to be a purely local reaction confined to the tissues as the reacting system, for no evidence of a systemic response has been seen. Previously Kraissl and Kasten had shown that of a series of patients with a history of burst abdomen, all were sensitive to catgut, whereas such sensitivity was only rarely found in patients without such a history. Also, these authors were able to show experimentally that burst abdomen following laparotomy was much more common in sensitized animals, and they suggested that if catgut was used in a patient in whom it was suspected that the wound might break down, the patient should be rapidly desensitized after operation with an extract of the catgut, or if that was not available, with sheep serum. It would seem obvious, however, that in such cases silk would be more efficacious as a suture material and would avoid the necessity for desensitization after operation. Still, those surgeons prejudiced against the use of silk are probably little influenced by this argument. If it were possible to determine beforehand which patients would develop a sensitivity to catgut and in which the danger of a wound disruption might arise, then there would be a strong argument against the use of catgut in such cases. Unfortunately, such sensitization is not usually manifest until after operation; thus pre-operative skin tests have no practical significance in determining the cases in which the catgut may be used with safety. In his series of thoracotomies Langston found that when a catgut sensitivity developed it was usually after the first or second stage, and that wound complications were more common in later stages than in earlier ones; that is, they were more common after sensitization. Apart from other reasons it would therefore be wise to avoid the use of catgut for the later stages of many staged operations. Various hypotheses have been advanced as to the antigenic agent in catgut, but it is probable that the obvious theory that this agent is the protein itself is the correct one.

In these columns the use of unabsorbable suture and ligature material has been discussed on a previous occasion (November 8, 1941, page 544), but in view of the increasing popularity of silk sutures and ligatures it is worth while briefly summarizing the rules that Halstead laid down in 1913 for the use of silk. These rules have been ignored by many surgeons and as a result the use of silk and other unabsorbable materials has fallen into disfavour with some people. A special advantage of silk and cotton at the present time is that they are much more readily available than catgut, and therefore it is urged that even the protagonists of the use of catgut should be familiar with the "silk technique" in case they are required to operate in an emergency in which supplies of catgut are not readily available. Halstead's rules are: (i) Very fine silk only should be used. (ii) Only interrupted sutures should be inserted. (iii) Many fine sutures should be used instead of a few larger ones if strength of the suture line is required. (iv) Complete hæmostasis must be secured. (v) Adequate skin preparation is required. (vi) Silk and catgut should not be used together in the same wound.

[1] *Annals of Surgery*, January, 1942.

LOSS OF A FINGER TIP.

IN these days of extensive injuries from automobile accidents and gunshot wounds the loss of a tip of a finger may be considered relatively unimportant and unworthy of prolonged treatment in an attempt to restore it. Nevertheless, the patient, especially if he is a typist, musician, electrical worker, skilled mechanic *et cetera*, may have different ideas on the subject and be willing to undergo a relatively long and tedious convalescence if there is a reasonable chance that the part will be restored to normal. This is all the truer if he has seen his colleagues handicapped by the loss of a finger tip and further disabled by the thin, easily ulcerating, painful scar which often results from the healing of wounds in this region. Often these wounds are closed by free grafts such as Thiersch, Thiersch-Ollier (split grafts), and full thickness grafts, by tube grafts from the skin of the chest or the abdomen, or by sleeve or pocket grafts from the abdomen, hip or other area; but the imperfect results usually obtained by these methods make the procedure recently described by R. A. Jones[1] worthy of consideration. This author recommends the use of a flap from the palm of the hand, which carries the underlying fat; for he states that in his experience it fills the defect more adequately than these other types of grafts. After careful cleansing of the hand and *débridement* under local anæsthesia the finger is flexed against the palm and a flap is turned up from the thenar or hypothenar eminences. It is most essential that the incisions should not cross the important creases of the hand, and also that the flap should be wide enough to cover the defect without tension. As much fat as necessary is raised with the skin. It is also recommended that the flap should be raised with the pedicle proximally or laterally, whichever is the more convenient. The finger is then approximated to the palm and the distal end of the graft is sutured to the skin between the defect and the nail, to the nail itself, or to the skin on the dorsum of the finger, depending on the nature of the wound. The sides of the graft are sutured to the defect as far proximally as possible without tension and without kinking of the graft. The finger is held in position after operation by adhesive tape. The sutures are removed about the tenth day and the adhesive tape is reapplied. After fourteen to eighteen days the graft is detached from the palm and the finger completely extended. A dressing is then applied with the finger slightly flexed till the wound heals.

The complications which might be expected to arise as a result of this procedure are disposed of by Jones. Firstly, there is no skin to spare in the palm of the hand and a scar in this region is often disabling. If, however, the wound is made on the thenar or hypothenar eminences the scar is mobile, non-painful and not disfiguring. In older individuals immobilization of a finger for two or more weeks might be expected to cause contracture of the joints of the finger. Jones asserts that in his cases by attention to detail during the operation and during the post-operative period this complication is not serious enough to offset the good features of the method. With the use of the finger, irregularities in the graft and scar tend to smooth out and later to disappear, so that Jones claims that it is difficult to see where the graft was placed. Sensation reappears in the graft within a few weeks. Jones reports that he observed no painful scars or neuromata.

Since the loss of a finger tip may cause considerable disability in certain groups of people and since the method of repair as described by Jones should give good results, it is to be recommended for trial in suitable cases.

[1] *The American Journal of Surgery*, February, 1942.

Abstracts from Medical Literature.

SURGERY.

A Method to Prevent Fresh Costal Cartilage Grafts from Warping.

G. B. NEW AND J. B. ERICH (*The American Journal of Surgery*, November, 1941) discuss the tendency of fresh cartilage grafts to warp after insertion, and describe a method of prevention. The basis of this method is the destruction of the elasticity of the cartilage by fixation with heat. The cartilage must not be boiled, and is most conveniently heated in a test tube immersed in a beaker of boiling water. Observations have shown that in such circumstances the temperature within the test tube does not approach more closely than to within 3° or 4° C. of boiling point. The authors advise filling the test tube with an aqueous solution of "Merthiolate". After heating for ten minutes, the cartilage is transferred to ice-cold normal saline solution for fifteen minutes. It is then ready to be shaped. The graft or any superfluous pieces of cartilage may be preserved indefinitely in an ice-cold aqueous solution of "Merthiolate".

Bile Peritonitis.

CHARLES W. MCLAUGHLIN, JUNIOR (*Annals of Surgery*, February, 1942), presents a series of eight cases of bile peritonitis. In the absence of obstruction, bile tends to take its natural course through the sphincter of Oddi and into the duodenum, even though there may be some abnormal opening in the duct system above the sphincter. Any such opening tends to close spontaneously, so that bile peritonitis is not a common condition. However, leakage may occur from a variety of conditions which the author classifies. These include rupture following abdominal trauma (though this is rare in the absence of previous biliary disease), slipping of a ligature from the cystic duct, and rupture of some portion of the duct system due to a rise of duct pressure when cholecystectomy has been performed and a stone in the common bile duct has been overlooked. Other causes are leakage from a divided and unrecognized accessory bile duct, and perforation, which may be gross or microscopic, following biliary infection. Bile peritonitis following gangrene of the gall-bladder is rare, as adhesion formation usually limits the process. Experimental studies suggest that the serious effects of this condition may be due to infection being carried into the peritoneum by infected bile, or to the bile so damaging the peritoneum that pathogenic organisms can pass through and determine peritonitis. In addition to these factors, there may arise a condition similar to surgical shock. The peritoneal cavity of laboratory animals with bile peritonitis becomes distended with bile-stained fluid. It is suggested that the extravasated bile injures the blood vessels, causing an increased permeability with a tremendous outpouring of plasma from the blood into the peritoneal cavity. This leads to hæmoconcentration, a fall of blood pressure, a rapid pulse rate and a clinical condition of shock. This alone may determine a fatal issue, and if not, it may lower the patient's resistance and allow infection to occur more readily. Therefore when a patient convalescent from an operation on the biliary tract develops a condition suggestive of delayed surgical shock, extravasation of bile should be suspected. When a drainage tube has been left *in situ*, the escape of bile will confirm the suspicion. When the abdomen has been closed completely or when the condition has arisen apart from operation, the diagnosis may not be suspected. The picture may simulate perforated peptic ulcer, or in the later stages ruptured appendicitis, general peritonitis, pancreatitis or intestinal obstruction. Coronary thrombosis has been suggested. The treatment involves drainage at the earliest possible moment. If found, the perforation should be closed, but this is rarely possible. Free bile and peritoneal fluid should be removed by suction. Drains are placed to the common duct area, and perhaps to the pelvis. Supportive treatment is necessary and replacement of fluid by plasma, if available, and by other parenterally administered fluids. The mortality rate is high, ranging from 50% to 75% in reported clinical cases. The mortality in the author's series was 62%.

Secondary or Post-Operative Parotitis.

GORDON F. MADDING AND ROBERT E. FRICKE (*Surgery*, January, 1942) report on a series of 190 cases of post-operative parotitis. Many theories of the causation of this condition have been advanced, but opinion today favours the theory of an ascending infection from the mouth along Stensen's duct. It has usually been thought that the mortality rate from post-operative parotitis is high, even as high as 60%. The authors contend that this is not so. In only one of their 190 cases was death due directly to the parotid infection. In the other seventeen fatal cases, there were other causal factors for the fatal termination. The diagnosis is usually easy. The onset occurs most often between the first and fifth days after operations on the colon, pelvis, biliary tract and stomach in that order of frequency. There are swelling, fever, and discomfort on jaw movements. The orifice of Stensen's duct is usually visibly inflamed. Gentle massage of the gland may express turbid fluid or pus. In rare cases, and usually only if suppuration occurs, the facial nerve may be involved. Prophylaxis consists of avoidance of dehydration and drugs which make the mouth dry, associated with careful oral hygiene. Treatment involves cold and hot packs, and sometimes air-spaced diathermy. Radium irradiation has been recommended, but the authors prefer X-ray irradiation. Surgical drainage is reserved for cases of suppurative parotitis. The presence of pus may be difficult to detect, as fluctuation may not be obtained under the firm fascial capsule of the gland. Aspiration may be useful when this difficulty arises, but the authors recommend that incisions be postponed until definite fluctuation is present.

The Undescended Testicle.

CHARLES E. REA (*Archives of Surgery*, January, 1942) has made a histological study of 46 undescended testes in patients past puberty, with the object of determining what should be done in these cases, and what the results of surgery are likely to be. The normal seminiferous tubule is lined by Sertoli (nourishing) cells, between which lie the spermatogonia or progenitors of the adult male sex cell. These spermatogonia pass through the stages of spermatocytes of the first order, secondary spermatocytes, and spermatids to become ultimately adult spermatozoa. In the 46 testes studied, only seventeen showed spermatogonia, three contained spermatids, and none showed spermatozoa. Speaking generally, the atrophy became more marked as the age of the patient advanced. The further the organ had descended, the more it tended to resemble the normally placed testis of the same age. Seven patients with bilateral cryptorchidism were treated by endocrinotherapy or operation or both. In one patient who received both types of treatment, spermatozoa, which had been absent from the semen prior to treatment, appeared in the semen some eighteen months later. In the other six cases, no spermatozoa were found in the semen after treatment. It seems to be agreed that the patients in whom the testis has failed to descend either spontaneously or as a result of endocrinotherapy by the time of puberty, should be subjected to operation. The author suggests that a functioning testis may be obtained after orchiopexy, even up to the third decade of life. Beyond the third decade, operation may be indicated for cosmetic reasons, but the function of the testis is unlikely to improve. But the testis is often small, and the cosmetic result in this case may not justify operation. If there is, however, an associated hernia, or if malignant disease of the testis is suspected, or if the organ is markedly shrunken and atrophied, orchidectomy should be performed.

Gas Gangrene.

H. J. WARTHEN (*Annals of Surgery*, April, 1942) discusses the incidence of gas gangrene in Virginia, reviews the results of various methods of treatment and suggests a method of management. Statistics suggest strongly that the virulence of gas-forming infections varies with time and locality, but are confusing when an attempt is made to evaluate the merits of the different therapeutic measures. No single drug or agent has proved specific. In the author's experience antitoxin has proved disappointing, and he feels unable to assess the value of prophylactic and therapeutic irradiation. He advises that every case in which the deep fascia has been penetrated should be regarded as a potential case of gas bacillus infection, and should be managed in the following way. A painstaking clean-up with sterile water, green soap and shaving is followed by the use of ether, iodine and alcohol. The operative field is draped, and a purposeful *débridement* is undertaken. Muscle removed is examined by culture methods for gas organisms. All parts of the wound are flooded with normal saline solution, the excess being dried out, and the wound packed with sulphanilamide. Anti-tetanic serum is administered, and if the surgeon has faith in it, gas gangrene antiserum. If the circulation in the part is impaired, an injection of "Novocain" to the sympathetic is advised. As soon as possible on the day of operation, a

radiograph is taken, partly to check the position of bone fragments, but chiefly to serve as a standard for comparison when later skiagrams are taken to determine the presence of gas in the tissues. A prophylactic irradiation may be given at this time. Twelve hours and twenty-four hours later further films are made. If both of these are "negative" the likelihood of the occurrence of a gas infection is small. Six grammes of sulphanilamide or sulphathiazole should be given daily for the first two or three days. The X-ray examination usually affords the earliest means of diagnosis, but a watch should be kept for excessive pain, swelling, tachycardia, discoloration or crepitus. If gas gangrene develops, an immediate *débridement* or amputation should be performed, depending on the degree of involvement and the state of the circulation. Irradiation and the use of sulphonamides should be continued. Blood counts should be made and transfusion carried out as necessary. An electrocardiographic examination should be carried out before the patient leaves hospital.

Symposium on Burns.

THE issue of the *Annals of Surgery* for June, 1942, includes a symposium on burns. V. E. Silor and M. R. Reid describe clinical and experimental observations on the use of "pressure" dressings on burns, and conclude that pressure over the burned area tends to reduce plasma loss, both at the surface and into the tissues.

W. E. Lee, W. A. Wolff, H. Saltoustall and J. E. Rhoads review recent trends in the treatment of burns. They endorse the value of plasma transfusions in the treatment of shock, and the use of sulphonamides both locally and generally in the prevention of infection. They stress the importance of maintaining the nutrition of patients with extensive burns.

H. N. Harkins discusses the local treatment of burns, and the importance of its careful correlation with general treatment. He considers that rapid tanning of the large flat surfaces of the trunk or proximal extremities seems the best method for wartime use. The face, hands, feet and genitalia should be given individual treatment such as sulphathiazole or sulphadiazine ointment. The author emphasizes the importance of early skin grafting in third degree burns.

Results of Thyreoidectomy.

L. DOBSON, H. SEELY AND H. ROSE (*Annals of Surgery*, February, 1942) discuss the results of a series of 341 consecutive operations performed at the clinic service of the Stanford University Hospital by various surgeons on patients suffering from diseases of the thyreoid gland. They pay particular attention to the post-operative complications and the final results. Pre-operative treatment and investigation were thorough, and operation was not performed until the patient showed improvement indicated by a decrease or disappearance of symptoms and a satisfactory and persistent lowering of pulse rate and of basal metabolic rate. In the operation described as having been used at this clinic the isthmus was divided first and the lobes were amputated from the medial to the lateral side. Two patients had wound infections, and several others developed hæmatomata and collections of serum requiring treatment. Eight patients had post-operative tetany. Six were mild cases, two were moderately severe, necessitating the administration of calcium by mouth and parathormone. One patient had severe tetany requiring parathormone and intravenous administration of calcium chloride. Two years after operation, this patient still needs "Viosterol" and calcium by mouth. Routine post-operative examination of the vocal cords revealed paralysis of one cord in each thirty cases; in at least seven cases the function of the cord returned after four to six months. One patient had bilateral temporary paralysis, function of both cords returning after six months. A few patients had temporary hoarseness, but in no case did any patient lose the voice or have respiratory difficulty. In this connexion the authors point out that a clear voice is no indication of normal vocal cord function. Interference can be determined only by laryngeal inspection. Follow-up investigations showed that nine patients had recurrence of toxic symptoms. Some were operated on again, but in the milder cases the patients were treated by exposure to X rays with complete success. The most satisfactory cases in the series were those in which the patients had a temporary hypothyreoidism after operation. The authors consider that the production of a hypothyreoid state is desirable in patients with coronary disease.

Arterial Embolectomy.

ARTERIAL embolism is defined (G. H. Pratt, *The American Journal of Surgery*, June, 1942) as the sudden, complete, shocking closure of a major vessel, usually previously normal, with the resultant threat to the life of the individual and the affected part. In 80% of cases there is left-sided heart disease, usually a rheumatic endocarditis, and the condition is often precipitated by some additional strain such as the onset of auricular fibrillation or a surgical operation or an acute infection. The onset occurs with sudden pain, shock and loss of function of the part. The limb becomes marble white and later blotchy blue. Characteristic colour changes of gangrene may follow. The part is cold and arterial pulsations are absent. The condition may usually be distinguished from arterial thrombosis occurring in an already diseased artery, by its sudden onset and the absence of previous signs and symptoms of partial obstruction to this or other portions of the arterial tree. In acute thrombophlebitis, the limb is warm and cyanosed and arterial pulsations are present. The embolus usually becomes impacted at the bifurcation of an artery, generally the common femoral, less commonly the common iliac, the popliteal, the brachial or the abdominal aorta. The author believes that the treatment should be surgical as soon as the diagnosis is made. The method has been developed mainly in the Scandinavian countries. Anæsthesia should be local, without the use of adrenaline. A tape is passed round either end of the exposed portion of the artery and the lumen is opened. The thrombus is gently removed from the distal portion of the artery first, and then the proximal portion is dealt with. Considerable ingenuity may have to be exercised in order to remove not only the thrombus which has formed locally, but also the embolus itself. The author has found a fine instrument like a corkscrew very useful. The arterial wound is closed with fine arterial silk on minute curved needles so placed as to avoid the intima. Over-suturing should be avoided and is unnecessary. Heparin may be used locally or post-operatively to reduce the tendency to clotting. The leg is not elevated, but is treated with great care to avoid injury and pressure effects. Vaso-dilators are valuable. A watch must be kept for further emboli which may need removal. Lumbar sympathetic nerve block may be helpful. Results are difficult to assess. Even a successful embolectomy will not prevent death from an accompanying severe heart lesion. But there seems no doubt that the death rate is much higher among patients not subjected to this treatment, owing to the onset of gangrene and the operations necessary for the treatment of this complication. The results of operation appear to approximate to the results obtained with operation for carcinoma of the breast in regard to survival after three, five and ten year periods, and operation for carcinoma of the breast is still considered to be worth doing. The author concludes by quoting a personal case in which he successfully removed an embolus at the aortic bifurcation, sixty hours after the disaster occurred.

Traumatic Cerebro-Spinal Rhinorrhœa.

KENNETH EDEN (*The British Journal of Surgery*, January, 1942) reports two cases of cerebro-spinal rhinorrhœa successfully treated by operative closure of the fistulous tract through which cerebro-spinal fluid passed into the nose. In the acute stages of head injury, leakage of this fluid through the nose is frequently seen, and in many cases the fistulæ heal spontaneously. But the fistula may persist, and when it does there is grave danger of a spread of infection to the meninges following catarrhal infections of the nasal sinuses. Lawson, in 1934, even went so far as to state that such cases are always fatal from secondary meningitis unless the fistula closes spontaneously or is closed by operation. In addition to the danger of meningitis, there is also at times a condition of intracranial aerocele with progressive increase of intracranial pressure. It is therefore recommended that operation should be undertaken in all persistent cases of cerebro-spinal rhinorrhœa. The diagnosis may be overlooked if the patient is not questioned about running from the nose, and tested by jugular compression, during which the flow of cerebro-spinal fluid may increase. In the two cases recorded, one of which was complicated by intracranial aerocele, operative approach was by a frontal osteoplastic flap. The fistula was closed by application of a fascial patch on the inner surface of the dura. The intradural method was chosen in order to avoid if possible some of the danger of infection which accompanies the use of the extradural route. The fistula frequently opens through a fracture into the frontal sinus, and disturbance of this area involves additional danger of a spread of infection leading either to sloughing of the fascial flap or to meningitis. The patients discussed were well eight months and two years after the operation.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on May 28, 1942, DR. W. F. SIMMONS, the President, in the chair.

Variations in Normal Heart Sounds.

DR. E. H. STOKES read a paper entitled "The Normal Heart and Conditions Simulating Heart Disease" (see page 253).

DR. D. G. MAITLAND thanked Dr. Stokes for his interesting paper. He said he wished to refer to the X-ray point of view with regard to children and the size and shape of the normal heart. Occasionally the radiologist was called upon to examine an infant with obvious clinical signs of congenital heart disease. It was a striking fact that there was often no change from the normal cardiac contour in such infants, because, if the cardiac lesion was compatible with life, it did not become apparent until it had produced secondary hypertrophy and dilatation in the chambers of the heart. It was then that the radiologist was able to see direct and indirect evidence of the congenital lesion. X-ray examination of the hearts of infants was of little use.

Dr. Maitland went on to say that the habitus of the patient played a great part in the shape of the normal heart, and had to be taken into account when the contour of the heart was being assessed. In the hyposthenic type of patient "dropped" heart was usual, and in the hypersthenic type the heart was more transversely placed. The radiologist was sometimes asked to examine a heart for any lesion that might be seen, and often all that was shown in the X-ray film of the heart of a patient manifesting, for example, anginal pain, was hypertrophy of the left ventricular shadow. Cases also occurred in which calcification and plaques were an incidental finding in the aortic area. Such persons were often apparently normal and had given no clinical sign of heart disease. With regard to conditions simulating heart disease, Dr. Stokes had mentioned gastric or duodenal ulcer. Dr. Maitland said that another very common coincident condition was cholecystitis.

DR. R. A. M. ALLEN also thanked Dr. Stokes for his paper. He said that for him the most interesting point of the paper was the emphasis that had been laid on the fact that a diagnosis of heart disease, or "bad heart" or "heart trouble", carelessly or unthinkingly made and communicated to the relatives, often led to great distress for the patient, a lifetime of assumed illness, and altogether a domestic régime that was not in the best interests of the persons concerned. Dr. Allen was reminded of a case of his own that had occurred many years earlier. The patient was a young woman, aged about twenty-three or twenty-four years, who was about to graduate at the university, but not in medicine. Her course had been somewhat delayed by a "bad heart". For that reason she had not been permitted by an anxious mother "to set foot on the earth". She was taken to the university by a motor car, which called for her and brought her home. Her height was about five feet and her weight about fourteen and a half stone. She had a mitral systolic murmur, but no other abnormality. Dr. Allen had created an absolute turmoil in the family when he insisted that the patient should walk at least a mile a day, to begin with. In the first fortnight she lost one stone in weight. A reduction in food intake and various other general measures brought down her weight still further. The last that Dr. Allen had heard of her was that she had returned from a trip abroad, which had included a walking tour through Ireland and another through the Austrian Tyrol, and was apparently enjoying life. There were no signs of disabling heart disease.

DR. W. J. MCCRISTAL thanked Dr. Stokes for his excellent presentation of the subject. He said that of those patients who came along with symptoms, apparent or real, referable to heart disease, there was in the first instance in a big percentage of cases no cardiac background and in the second instance unnecessary invalidism had been imposed, and in either case probably the foundation for a cardiac neurosis had been established. Then again one was not impressed with the value of hospital records and statistics when through clumsiness or incompetence inaccurate designation of a condition was applied. A recoverable myocarditis was mistermed myocardial degeneration. The bizarre cardiovascular syndrome of the psychoneuroses was confused with the more legitimate effort syndrome to be found in the neurasthenias. After Crile's experiments the latter was a physical state and the anxiety neurosis a psychogenic state.

Dr. McCristal said he was not quite sure that Dr. Stokes had made or was prepared to allow this distinction which he regarded as important in the handling of these cases in which army wastage, invalidism and pensioning schemes were involved. Dr. Stokes had observed that the subject of the paper was of national importance, and Dr. McCristal said that because of this and the foregoing remarks he proposed to be somewhat provocative and to suggest that more care should be exercised in the allocation of medical officers to boards in cardiac cases and their more involved implications. Dr. McCristal said that one had only to recall Lewis's figures of the last war, in which of 70,000 men invalided by the end of 1918 as suffering from heart disease, only one out of every six were subsequently proved to have heart disease. The others suffered from conditions which comprised neuro-circulatory asthenia, with or without systemic disease, anxiety states *et cetera*. In conclusion, Dr. McCristal thanked Dr. Stokes sincerely for his paper and expressed regret that more members were not present to hear it.

DR. DOUGLAS ANDERSON said that no speaker had alluded to Dr. Stokes's records of heart sounds. Surely history had been made at that meeting. Dr. Stokes had given no names to the records, and Dr. Anderson wondered whether "cardiophony" would do. Dr. Stokes had said that he began the making of such records in an unambitious way, but that he proposed to make further records and use them for teaching purposes in the future. Dr. Anderson had been caused by the records to think back thirteen or fourteen years to the first talking pictures heard in Sydney and the sounds of Al Jolson, which had been replaced by others till at the present time one heard the dulcet tones of Clark Gable. Dr. Anderson wondered what was in store for audiences in the future. He thought that Dr. Stokes was to be congratulated on the new method, and he wished him success. Dr. Anderson went on to say that most speakers had alluded to those patients who presented themselves with symptoms, principally pain and a sense of constriction in the chest—symptoms that might be referable to the heart in their own minds—and with other symptoms that the doctor might suspect of being due to cardiac disease, such as dyspnœa. In such cases it was important to distinguish between real dyspnœa due to anoxia or to pulmonary or cardiac causes, and that other feeling which was not dyspnœa, the inability to draw a satisfying breath. The last-mentioned was the leading symptom of many persons suffering from the various psychoneuroses. Such patients, in spite of all their symptoms, might present no abnormality of the heart according to all the criteria set out by Dr. Stokes; they presented a problem in treatment. Dr. Anderson uttered a plea that doctors dealing with such people should spend a great deal more time in hearing what the patient had to say—not so much from the point of view of his symptoms as from that of the background of his everyday life. In that way, and in that way only, would the background of his disability be elicited. In the treatment of such people reassurance was not enough. The patient might be relieved in mind, but scarcely convinced, when he was assured that his heart was normal, that no abnormality could be found. Only too often his symptoms persisted and he frequented the out-patient department month after month or visited doctor after doctor. Dr. Anderson suggested that such people might be greatly assisted, if not relieved of their symptoms, by a fuller explanation of what might appear to be the psychological background elicited from their history.

DR. F. A. E. LAWES thanked Dr. Stokes for his able presentation of a difficult subject. Dr. Lawes referred to the symptom of pain in the chest that was encountered commonly in general practice. Dr. Lawes had been asked by a surgical colleague to examine a middle-aged man suffering from pain in the chest; the surgeon wished to know whether it was due to coronary disease, and asked Dr. Lawes to carry out a full examination, if necessary with electrocardiography. Dr. Lawes remembered that such pain was often due to fibrositis, and that it was not substernal, and that it was frequently possible to find the tender area. He had on many occasions been aided by Lewis's statement that if the pain was anginal it would always follow exactly the same amount of exercise. The pain of fibrositis was extremely variable and would occur after every grade of exercise. The patient under consideration was aged thirty-six years, and had been working for fourteen hours a day in a munition factory. When Dr. Lawes's surgical friend had examined him, the apex beat was five inches from the mid-sternal line and the pulse rate was 120 per minute. By the time he went to Dr. Lawes for examination he had had rest

and the pulse rate had dropped to 72 per minute; the heart was then not enlarged. The patient had been working very hard, had had several attacks of giddiness and was afraid that he was losing control of himself. Dr. Lawes put him through an exercise tolerance test and found his heart perfectly normal. He was suffering from sepsis due to infected teeth and from an inadequate intake of vitamin *B*; but his chief trouble was exhaustion. Dr. Lawes had no hesitation in assuring his surgical colleague that that was the whole trouble, and no cardiographic examination was necessary.

DR. R. S. STEEL said that Dr. Stokes had presented a difficult subject in a very explicit manner. One thing had pleased Dr. Steel greatly; that was Dr. Stokes's insistence that electrocardiographic tracings were to be considered in conjunction with the clinical history and findings. Dr. Steel had taught that over and over again, but he was afraid that a diagnosis was often made from tracings. The man who interpreted the tracing gave his viewpoint from what he had learnt from books and perhaps from his own clinical experience; but in the majority of cases he did not examine the patient. Psychological factors might affect a normal heart tracing. Another point to which Dr. Steel referred was the loud, booming first sound heard at the mitral area in large, muscular, athletic subjects. Dr. Steel did not wish to mention "athlete's heart", because there was no such thing, except that it was a large heart. In the type of person to which he was referring there was a loud, booming ringing sound. Dr. Steel recalled a first grade football player for the university, who was under the penalty of being classed as a "cardiac" subject for life on the grounds of a systolic murmur. Dr. Steel examined the man's heart and found this loud, booming, ringing sound, which he had often found in large, muscular men. It was probably due to slight hypertrophy of the heart causing an extra bound of the heart on the chest wall. It was not to be mistaken for a systolic murmur or even for a presystolic murmur. Dr. Steel went on to say that there was another university footballer at the present time with an electrocardiographic tracing that appeared an absolutely crazy thing; everything in it appeared wrong. The question arose as to whether the young man should be left out of the football team. In 1941 he played football every week. At the present time he was practising. So far there was nothing wrong with him clinically. These were further cases in which it was essential to correlate the clinical findings with abnormalities found in the electrocardiographic tracings. Dr. Steel asked Dr. Stokes in his reply to refer to such conditions.

DR. H. J. DALY added his thanks to Dr. Stokes to those expressed by earlier speakers. He said that in Levine's book "Clinical Heart Disease" the author laid great stress on the fact that the vital capacity of a patient was of importance in the differential diagnosis of neuro-circulatory asthenia from organic heart disease. Dr. Daly was very interested in Dr. Stokes's records of cardiac sounds; he asked whether they could be slowed at all. He thought it would be possible to analyse them much better if the record could be played slowly.

Dr. R. A. M. Allen asked Dr. Stokes in his reply to allude to the fact that before the last world war a mitral systolic murmur was regarded as evidence of definite cardiac disease. Then it was found during the war that a great number of really physically fit young men had mitral systolic murmurs. A swing of the pendulum followed, and mitral systolic murmurs were regarded as being of no significance whatever. Dr. Allen understood that at present a return of the pendulum was occurring, and mitral systolic murmurs were being assessed in the light of the clinical findings, history *et cetera*.

Dr. Stokes, in reply, thanked those who had taken part in the discussion for the way in which they had received his paper. Dr. Maitland had referred to the confusion of cholecystitis with cardiac disease. Dr. Stokes endorsed his remarks. He pointed out that one of the difficulties was that cholecystitis and organic heart disease were often associated. Only the previous week he had had a patient who had undergone operation for cholecystitis; this patient also had bundle branch block, but she had withstood the operation quite satisfactorily. Dr. Allen's reference to the diagnosis of heart disease when none existed was interesting; such cases were not infrequent. Dr. Stokes had recently had referred to him a girl who was thought to have heart disease. She gave a vague history of rheumatism. Dr. Stokes had put her through all the tests, and had found her heart quite sound. The girl's guardian would not let her play games. It was proposed that she should go to Kosciusko, and after some difficulty she was allowed to go; the trip and the exercise did her good. With regard to Dr. Allen's remarks on the significance of mitral systolic murmurs, Dr. Stokes said that he agreed that a swing of the pendulum seemed to be taking place. In such cases he thought that the heart should be auscultated with the patient in all positions; functional murmurs disappeared when the patient was erect. The size of the heart varied greatly with alterations in posture and in different phases of respiration. Dr. Stokes again referred to a French expedition which some years earlier had gone to India and had examined the Yogis. What particularly impressed the members of the expedition was the fact that great alterations occurred in the electrocardiograms. Dr. Stokes reiterated his suggestion that such alterations might have occurred as a result of the subject's control of his diaphragm. Dr. McCristal had referred to effort syndrome. That term had first been used by Lewis; he had found that the symptoms arose after effort. Later investigation showed that they arose quite apart from effort. In all groups of conditions labelled neuro-circulatory asthenia, there was a large psychoneurotic element; it might be unconscious motivation. Lewis had done great work during the last world war; 80% of men with alleged cardiovascular disease were suffering from conditions such as effort syndrome. Dr. Stokes suggested that the work of Lewis and the Goulstonian Lectures of Wood should be widely read. Dr. Stokes next thanked Dr. Anderson for his remarks about the records of heart sounds. He said that so far they had been able to collect only a few cases, but he hoped as time went on to record normal heart sounds, normal variations in heart sounds and heart sounds indicating organic disease. Dr. Daly had asked whether the records could be played more slowly. Dr. Stokes had discussed the possibility with technical advisers from Amalgamated Wireless (Australasia) Limited. They considered that if the records were slowed the sounds would be altogether changed. Dr. Stokes agreed that it would be good to slow the records, as Dr. Daly suggested, and attempts would be made to do this. It was a difficult matter to reproduce heart sounds, because there was so much interference from various causes. Dr. Stokes expressed his gratitude to Mr. Brooker, who had worked with him to produce the records, and to Mr. Giles, who had also accompanied him to the meeting to play the records. Dr. Stokes had suggested the idea to Mr. Hooke some time ago. A start had been made two years earlier; but it was not until Dr. Stokes had been asked to read the paper that the production of a record had been attempted. Dr. Anderson had referred to the psychological background of patients presenting cardiac symptoms in the absence of any cardiac disease, and to the inadequacy of reassurance as a therapeutic measure. Dr. Stokes quite agreed with him; some of these people one could never make well. In the words of the immortal Osler, one "would have to rebuild them from the blastoderm". Dr. Anderson had said that these patients returned month after month to the out-patient department; when Dr. Anderson had been an out-patient physician for as long as Dr. Stokes, he would rather say decade after decade. Dr. Stokes was in agreement with Dr. Lawes's remarks on fibrositis; it was undoubtedly a frequent cause of pain in the chest. It had an unfortunate habit of cropping up at odd times, apparently with changes in the weather *et cetera*. Dr. Stokes agreed with Dr. Lawes's summing up of the case of the exhausted munition worker. Dr. Steel had referred to the large, muscular men with a booming sound at the mitral area. Dr. Stokes harked back to what he had said earlier, that the signs of disease were not as a rule isolated; there were usually several. The sound might be, as Dr. Steel had suggested, an indication of left ventricular hypertrophy. The case of the footballer with the queer electrocardiogram was difficult to interpret. But one should not be swayed, as Lewis had said, by such a thing, if other findings were normal. Dr. Stokes was in agreement with Dr. Daly that the vital capacity was not impaired as a rule in neuro-circulatory asthenia, but was impaired frequently in heart disease. He thought that that consideration might be an additional help in diagnosis, but there were other means at their disposal—the makeup of the patient, the history *et cetera*. In conclusion, Dr. Stokes stressed once again the importance of remembering the influence of posture and of respiration on the normal heart.

DR. W. F. SIMMONS, from the chair, on behalf of the members of the Branch present and not present, thanked Dr. Stokes for his interesting, instructive and valuable contribution to an important subject. As President, Dr. Simmons regretted the absence of a large number of members from the meeting. The committee which drew up the programme and included that subject, asking Dr. Stokes to discuss it, had expected that the hall would be full. As one who had known Dr. Stokes for over twenty years and had

been aware of his painstaking and thorough ways, Dr. Simmons felt that they had chosen the right man to speak on the subject. He particularly regretted the absence of those men and women who were interested in the Service side of the work. He expressed the hope that at any future meetings there would be adequate representation from members serving in the defence forces. Dr. Simmons agreed with Dr. Anderson that those present were under a debt of gratitude for the record of heart sounds presented; he had felt that such an innovation might be introduced by Dr. Stokes. Dr. Simmons knew the thoroughness with which Dr. Stokes took up every request to be the speaker at a Branch meeting. Dr. Simmons had learned a great deal from Dr. Stokes's paper. He himself did a considerable amount of medical "boarding", and from that point of view, and also in his capacity as a general practitioner, he thought that the paper would be of great value to him throughout the rest of his career in the assessment of the normal and abnormal heart.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 18, 1942, at the Royal North Shore Hospital of Sydney. The meeting took the form of a number of clinical demonstrations by the members of the honorary medical staff of the hospital.

Colles's Fracture.

DR. A. R. HAMILTON showed several patients to illustrate methods of splinting Colles's fracture. In the first case the fracture had been treated by Robert Jones's method. Dr. Hamilton pointed out three features: (i) the use of spiral aluminium splints, the dorsal splint spiralling in the lower part towards the head of the radius; (ii) the use of a short volar splint to the fracture line only, with a pad on the end; (iii) the dorsal splint extending down to the level of the metatarsal heads, a pad being placed over the distal fragment of the radius.

Dr. Hamilton's second patient was a male, aged ten years, who had sustained a Smith's fracture (reversed Colles's fracture). The fracture had been sustained on May 27, 1942, and reduced two days later under fluoroscopic control. Dr. Hamilton pointed out that the arm was put in full supination, in order to obtain purchase on the displaced radial fragment and so prevent recurrence of the deformity. A plaster cast extended above the elbow to prevent rotation of the forearm.

The third patient shown by Dr. Hamilton was a female, aged fifty-six years, who had sustained a comminuted Colles's fracture on June 7, 1942; an attempt had been made to reduce it on the same day, but X-ray examination showed the position to be unsatisfactory. On June 12 the fracture was reduced under fluoroscopic control. Dr. Hamilton pointed out that the limb had not been put in full palmar flexion because of the danger of post-traumatic arthritis, and so that the volar fragment would be held in alignment. Full ulnar deviation was required to maintain the length of the radius. A circular plaster cast was applied and then bivalved for better fitting and to eliminate rough edges. The elbow was fixed with a short plaster arm slab to prevent movements of supination and pronation.

Osteomyelitis.

DR. E. D. CLARK showed a male patient, aged thirteen years, who had been admitted to hospital on March 24, 1942. He had been well until six days previously, when he noticed a pricking sensation in the region of the right hip. Later pain developed, radiating to the right groin; it was accompanied by loss of sleep and a high temperature. The symptoms gradually became worse.

On the child's admission to hospital, signs of fever suggested acute osteomyelitis of the ilium. The pain was most severe in the region of the anterior superior iliac spine. His temperature was 103° F. and his pulse rate was 112 per minute. Exploration of the iliac crest and the region of the anterior superior iliac spine was undertaken under anæsthesia. Osteotomy of the ilium revealed no sign of pus. The wound was closed and a drainage tube was inserted. The child's temperature remained high after operation, but occasional remissions occurred. Frequent X-ray examinations were subsequently made. At first no abnormalities were detected; but on April 5 evidence of osteomyelitis was detected on the iliac crest extending downwards over the ilium. A second osteotomy was performed and exploration was carried out over a wide area; there was no sign of pus. The wound was packed with "Vaseline" gauze and the hip was placed in a plaster cast, with closed drainage. A high degree of pyrexia continued, but the patient began to feel more comfortable. He was examined by Dr. A. R. Hamilton, who suggested more complete immobilization in plaster of Paris. When the cast was removed on May 20, massive granulation of the wound and a fairly copious discharge of pus had taken place. Dr. Clark said that since that date the patient had shown gradual signs of improvement, accompanied by slow remission of the temperature. At the time of the meeting he was almost afebrile. Sulphanilamide therapy had been carried out for various periods since his admission to hospital, but with no appreciable benefit. Dr. Clark remarked that the case was of interest, since in the early stages of the illness there was no sign of pus formation; this became evident to a slight extent later. Radiological examination revealed an increasing degree of osteomyelitis, which extended to the surrounding area of the hip joint; the joint was not affected. Slow improvement followed the application of a closed plaster cast. Chemotherapy was of doubtful value only.

Ruptured Uterus.

DR. OSSIAN ROBERTSON showed a female patient, aged twenty-six years, who had been admitted to hospital in labour on March 2, 1942. A male child weighing eight pounds was delivered normally twelve hours later. The patient had had one child four years previously. The puerperium was normal, and she was discharged from hospital on the eleventh day; the uterus was then in good position, but slightly subinvoluted. She was readmitted to hospital on April 4, on account of moderately severe hæmorrhage. After seven days' rest in bed and treatment by douches *et cetera*, all hæmorrhage ceased and she was discharged from hospital. She was readmitted on April 22 with further hæmorrhage.

On April 24 the uterus was explored under anæsthesia. A sound passed through the cervix met with no resistance and entered the abdominal cavity. The abdomen was opened, and the uterus was found to be split in the mid-line from the internal os to the fundus. The opening was found to be plugged with omentum and the raw edges of the tear had become covered with peritoneum. Hysterectomy was performed, and the patient made an uninterrupted recovery and was discharged from hospital on May 13. Dr. Robertson said that the rupture must have started during labour and been gradual, as there were no symptoms of shock or internal hæmorrhage.

Toxæmia of Pregnancy.

Dr. Robertson's second patient was a female, aged thirty-three years. She had had no previous illnesses and her general health had been normal until her first pregnancy in 1934. On that occasion she was well until the fifth month, when she began to suffer from swelling of the feet, arms and face. The blood pressure was 160 millimetres of mercury systolic and 110 diastolic for one month before the œdema was noticed. The urine was clear; the blood urea level was 17 milligrammes *per centum* and the blood creatinine content was 1·25 milligrammes *per centum*. She was admitted to hospital at seven and a half months; at that time generalized œdema was present, the blood pressure was 180 millimetres of mercury systolic and 100 diastolic, and the urine was almost solid with albumin. A classical Cæsarean section was performed when no improvement had been brought about by medical treatment. The baby, which weighed three pounds three ounces at birth, lived for two days. The patient's recovery was gradual, till after two years her health was as before.

When she became pregnant for the second time, the patient felt well for the first two and a half months, except for morning sickness and leg pains. At that time her blood pressure was 170 millimetres of mercury systolic and 100 diastolic, and her urine was clear. The urea concentration test revealed impaired renal function of moderate degree. At four months she gradually became dyspnœic on exertion; œdema, palpitation and visual disturbances followed. At six months she had œdema of the hands and feet, and the urine was three-quarters solid with albumin. The systolic blood pressure was 190 millimetres of mercury and the fundi were normal.

Dr. Robertson said that the patient, who was an excellent type of woman, evinced a keen desire to have a child. This was her last and only chance of doing so. It was therefore decided to allow the pregnancy to proceed till the fœtus reached a viable age, and then to perform Cæsarean section and sterilization. She was admitted to hospital on March 30, 1942, at six months' gestation. She was given complete bed

rest and a diet of fluids only for forty-eight hours, and then her intake of protein and salt was limited. These measures brought about the disappearance of the œdema and a reduction in the amount of albumin in the urine to a cloud, or on occasions none at all; moreover, the blood pressure fell from 225 millimetres of mercury systolic and 140 diastolic (the highest reading) to 190 millimetres of mercury systolic and 120 diastolic. No casts were found in the urine, and the blood urea and creatinine contents did not rise above 32 and 1·3 milligrammes *per centum* respectively. The fundi were normal.

As the eighth month of pregnancy approached, the blood pressure rarely fell below 200 millimetres of mercury systolic and 120 diastolic; headache and dizziness occurred, and the urine persistently contained a heavy cloud of albumin. On the day when the pregnancy reached eight months, the patient had abdominal pain and considerable hæmorrhage *per vaginam*. Cæsarean section was immediately performed; some retroplacental hæmorrhages and a small intramural hæmorrhage were present, and a living female infant was delivered. Apart from oliguria and albuminuria for a few days afterwards, the patient's convalescence was rapid and uneventful. The systolic blood pressure remained at about 150 millimetres of mercury; the urine was clear. On June 17 the blood urea content and the blood creatinine content were 33 milligrammes and 0·91 milligramme *per centum* respectively. The baby weighed three pounds at birth; she regained her birth weight in fourteen days, and on June 16, at the age of three weeks, she weighed three pounds five ounces.

Dr. Robertson said that the case illustrated toxæmia of a first pregnancy in which hypertension was the first feature, followed by dyspnœa, œdema and albuminuria, and toxæmia of a subsequent pregnancy, in which these symptoms occurred earlier, and which terminated in revealed and concealed accidental hæmorrhage. He concluded by paying a tribute to the courage, resolution and strength of will of the patient.

Squamous Carcinoma of the External Auditory Meatus.

DR. E. P. BLASHKI showed a male patient, aged forty years, who had been admitted to hospital on July 31, 1941; for several years he had been undergoing treatment to his right ear at various hospitals. The presenting symptoms were pain and a blood-stained discharge. A provisional diagnosis of epithelioma of the external auditory meatus was made. Examination of a biopsy specimen revealed squamous carcinoma; cell nests were present in a fibrocellular stroma. Infiltration had not extended to the cartilage in the sections examined.

On August 1 a radical mastoid operation was performed. The bone was sclerotic and the antrum was filled with foul pus. Pathological examination of the excised tissue confirmed the findings in the previous sections. Seven days after operation fourteen milligrammes of radium were inserted in the following manner: two amounts of two milligrammes in the upper edge of the wound parallel to each other, one amount of two milligrammes in the lower edge of the wound parallel to the upper two, and four amounts of two milligrammes in rubber tubes in the mastoid cavity. The radium was left *in situ* for ninety-six hours.

The patient's subsequent history was uneventful. The discharge was controlled by saline irrigations, and the wound was kept open. On May 8, 1942, a plastic operation was performed to close the retroauricular wound. A small plastic procedure was necessary also on the pinna, because the auditory meatus had become stenosed and an artificial opening had appeared above it. Small skin grafts, taken from the thigh, were applied to the auditory canal. At the time of the meeting the retroauricular wound had healed and the canal had nearly healed. A few small sequestra remained to separate from the middle ear area. No growth was to be seen.

Basal-Cell Carcinoma of the Antrum and Middle Ear.

Dr. Blashki next showed a female patient, aged thirty-six years, who had first been examined in October, 1938, on account of a blood-stained nasal discharge and nasal obstruction of eighteen months' duration. Examination of a biopsy specimen from the left nasal cavity revealed basal-cell carcinoma. The degree of malignancy was difficult to state, but the pathologist considered that local recurrence was almost certain. A radical antrostomy with exploration of the ethmoid cells was performed. All were filled with carcinomatous material. Pathological examination confirmed the diagnosis. Radium was inserted; the dosage was 2,088 milligramme hours, and one radon seed was implanted in the left nasal cavity. Subsequently a considerable amount of bony necrosis occurred, involving most of the antral wall; but no local recurrence was observed.

In May, 1942, the patient was again examined. She complained of deafness in and discharge from the left ear, of indefinite duration. On examination, the aural canal was filled with bleeding granulomatous material. Pathological examination revealed only papilloma and granuloma. The appearance subsequently suggested that the middle ear was filled with a tumour bulging the drum membrane.

On May 29 a radical mastoid operation was performed. The middle ear was found to be filled with soft, granulomatous material. Pathological examination revealed that the tumour was a basal-cell carcinoma, resembling the former tumour in the antrum. Radium was inserted five days later, in the following dosage: five milligrammes in the post-nasal space, embedded in dental "Stent", five milligrammes in the middle ear area, and two milligrammes in the wound near the flaps. The radium was left *in situ* for forty-eight hours.

Foreign Body in a Bronchus.

Dr. Blashki next showed a male patient, aged forty-one years, who had been admitted to another hospital with a cough and bronchopneumonia. He had taken part in a fight about two weeks previously, and portion of the alveolar margin of his denture was subsequently discovered to be missing. X-ray examination revealed bronchopneumonia of the left lung. At the time of his transfer to the Royal North Shore Hospital he had been ill for about six weeks. His symptoms were cough and sputum, sweating, and a rise of temperature to 101° F.; he had lost two stone in weight.

On May 8 a bronchoscopic examination was made under local anæsthesia with cocaine and general anæsthesia induced by the intravenous injection of "Pentothal Sodium". A foreign body was found in the left main bronchus, wedged and surrounded with granulations; it was removed by forceps with the use of the bronchoscope. After-treatment was undertaken by Dr. Bruce White; the patient was given sulphapyridine and short-wave therapy. His progress was uneventful, except for the occurrence of an iodide rash on one occasion.

Frontal and Maxillary Sinusitis.

Dr. Blashki's next patient was a male, aged twenty-five years, who had had severe frontal headaches for one year. He had been referred by another specialist for a radical operation on the right frontal sinus. His complaint appeared to date from a dental extraction one year earlier. He had an offensive, one-sided nasal discharge. The decision was made to perform a radical operation on the antrum for the dental infection, and to employ Harmer's operation for ventilation of the frontal sinus. On January 9, 1942, a Caldwell-Luc operation was performed on the right antrum, and Harmer's intubation operation was performed on the right frontal sinus. Dilatation of the frontal duct was continued until January 27, when there were three rubber bands *in situ*. With lavage and short-wave therapy the symptoms abated in about one month.

Dr. Blashki said that the patient was shown to illustrate the necessity of selecting and applying the means of cure to suit individual patients, particularly in suspected disease of the frontal sinuses. The case also showed that in the treatment of the frontal sinusitis, patience was rewarded.

Chronic Poisoning in a Child.

DR. DOUGLAS ANDERSON showed a male patient, aged six years, who suffered from weakness of the anterior tibial muscles and thigh muscles and from severe normochromic anæmia; both conditions had much decreased at the time of the meeting. X-ray examination revealed increased density of the ends of the long bones. Punctate basophilia was always to be found in several thousand erythrocytes per cubic millimetre of blood, and the child excreted relatively large amounts of porphyrin in the urine. He was undersized for his age, but alert and active. He had been brought to the hospital on January 31, 1942, on account of vomiting and pain in the right side of the abdomen. Foot drop was present. The erythrocytes numbered 3,500,000 per cubic millimetre of blood, the hæmoglobin value was 8·5 grammes *per centum*, the colour index was 0·82, the mean corpuscular hæmoglobin content was 24·2 and stippled cells numbered 3,800 per cubic millimetre. Lead poisoning was suspected; the child was found to excrete lead in the urine, but not in abnormal quantities. No likely source of lead poisoning could be found.

Dr. Anderson said that the child had been in another hospital in 1939 in a similar condition, and his recovery was incomplete. In 1938 he had had an illness called gastric influenza, and had always been inclined to vomit afterwards. Dr. Anderson pointed out that the group of muscles that were weak was one which was always spared in lead poisoning. In August, 1941, the erythrocytes numbered 4,600,000 per cubic millimetre of blood, the hæmoglobin value was 11·2 grammes *per centum* and the colour index was 0·86; no punctate basophilia was noted. Dr. Anderson invited suggestions for further investigation and treatment.

Cerebro-spinal Syphilis.

Dr. Anderson also showed a male patient, aged fifty years, who exhibited several signs which together were suggestive of cerebral syphilis; they were a particular type of tremulousness of the face, a coarse tremor of the tongue, an oval shape of the pupil and some mental hebetude. In addition he had a feeling of heaviness, tremulousness of the left hand, flutter of the eyelids, sluggishness of the pupillary reflexes, analgesia of the legs and feet, of the front of the chest and of the ulnar border of the right forearm, loss of the knee and ankle jerks (but not of all posterior column sensibility) and psoriasiform lesions on the lower part of the legs. He did not complain of headache, vomiting or impairment of vision. The brown coat on his tongue suggested hepatic damage or intoxication as well as focal cerebro-spinal disease. Dr. Anderson said that the tremors first mentioned as being suggestive of syphilis were also seen in some toxic psychoses, such as *delirium tremens*; but no such symptoms and outward signs of cerebral syphilis were characteristic, since they resulted from vascular or lymphatic damage or degeneration, which could be produced by such morbid processes. Both the blood serum and the cerebro-spinal fluid reacted to the Wassermann test; but the cerebro-spinal fluid was not characterized by pleocytosis or by an excessive content of protein.

Dr. Anderson said that the patient had received treatment at another hospital, but had ceased to attend there three years earlier, remaining well for one year and then being awarded the invalid pension on account of the condition of his nerves. The manual tremor developed suddenly one year prior to the meeting. It was hard to predict how much symptomatic benefit would result from the resumption of treatment. It could be said that symptoms due to cerebro-spinal hæmorrhages, degeneration or softening resulting from vascular damage would not be influenced by anti-syphilitic medication.

Raynaud's Phenomenon.

Dr. Anderson's third patient, a man, aged twenty years, suffered from aching and a heavy feeling in both forearms when he attempted to work and when he allowed the arms to hang by his sides. The hands were white and cold and the nails were cyanotic in even moderately cold weather. Immersion of the hands in cold water greatly aggravated the condition and caused aching; but when the ulnar nerve was blocked with procaine, its area of distribution remained pink and warm despite immersion of the hand in cold water. A drop of the patient's blood taken on a cold glass slide did not agglutinate or form rouleaux. The erythrocyte sedimentation rate at room temperature was normal. The patient did not appear to be neurotic.

Dr. Anderson went on to describe the patient's history in detail. For twelve months prior to the meeting he had been subject to a vague aching in both wrist joints when he attempted to work with his hands, and this aching travelled into the forearms, which felt intensely heavy. He was well when he did not work, and he could sometimes work for a day or two. Recently typing seemed to bring on his trouble, as did hanging the hands down. At the time of the meeting he was a professional musician, playing the saxophone, cornet and piano accordion. He had no disability in the legs or in other joints; neither had he any sweating, thumping of the heart, giddiness, headache, tiredness in the mornings or other symptoms of effort syndrome. He had no distaste for fatty food and no insomnia. Three years earlier, when he was doing heavy work in a hot "Bakelite" factory, he had a transitory painful swelling of both wrist joints. This subsequently recurred when he put a strain on his wrists. In the autumn of 1941 he noticed that cold weather or a cold bath made the hands very cold and heavy and caused aching in the wrists. Tight wristbands on a shirt or jumper or a wrist watch also caused aching. The summer, contrary to his expectations, brought no relief. He found that if he worked despite the symptoms they persisted for some weeks after he had ceased work. His feet were unaffected, and he did not suffer from chilblains. No numbness or paræsthesia occurred during the attacks. Sometimes he noticed "pins and needles" as the attack passed off. His father and mother were alive and well; they were both Austrian and not Jewish. The patient did not closely resemble either parent. Two brothers died soon after birth. The patient did not smoke, and took alcohol very seldom. He had had his tonsils and adenoids removed at the age of six years; appendicectomy had been performed when he was eleven years old; it followed a blow an the abdomen, and blood was said to have been present in the appendix.

On examination, the patient appeared well. His weight was eleven stone two and a half pounds, and his tongue was clean and moist. No abnormalities were detected in the heart, chest, abdomen, nervous system or wrist joints. The hands were white and the nails cyanotic. It was a cold day, but other parts were warm. Immersion of the hands in cold water aggravated their condition and caused aching; immersion in warm water relieved the condition, but caused little reactive hyperæmia. Procaine was injected into the right ulnar nerve at the wrist and then the hand was immersed in cold water; the ulnar nerve distribution remained pink and warm. A drop of blood taken on a cold glass slide showed no clumping or rouleaux. The erythrocyte sedimentation rate was seven millimetres per hour. On June 10 the serum calcium content was 9·8 milligrammes *per centum*; on June 12 serological tests for syphilis yielded no reaction. The provisional diagnosis was Raynaud's syndrome.

A Pleasantly Tasting Opaque Bolus.

Dr. Anderson finally described a modification of a pleasantly tasting opaque bolus, which had been shown at a meeting in 1938. As marmalade was in short supply, the bolus had been altered by the substitution of quince jelly. This had been found to be an improvement. The bolus consisted of a mixture of pure barium sulphate and quince jelly.

Hæmorrhage into the Sheath of the Optic Nerve: Optic Atrophy.

DR. W. MACDONALD showed a female patient who had suddenly lost the vision in her left eye six years earlier. No improvement in vision had occurred in the intervening period. Examination of the eye disclosed that the vessels on the disk were represented by small vestiges in the central portion of the disk. In the retina complete obliteration of the arteries and veins had occurred; the vessels were represented as fine, whitish threads. The disk was completely atrophied, and considerable pigmentation was present in the retina. Dr. MacDonald said that the only condition which would so completely cut off the vessels was hæmorrhage into the sheath of the optic nerve; he thought that that was what had occurred. The suddenness of the onset pointed to a vascular lesion.

(To be continued.)

Correspondence.

FOCAL INFECTION.

SIR: If Dr. Shallard's statement (editorial, THE MEDICAL JOURNAL OF AUSTRALIA, July 11, 1942; Dr. Kinsella's critical letter, July 18, 1942) that "focal sepsis has never been conclusively shown to produce anything but focal sepsis" is accepted, no single sentence could condemn more individuals to invalidism and, often in the end, to premature death.

Thus a robust and successful athlete, at the age of thirty, had two teeth devitalized under protest. For the next ten years he had progressive failure of health. His illnesses, which culminated in a toxic and nervous breakdown, included diphtheria at thirty-four years of age, and multiple arthritis with resignation from work. The teeth were ignored in all medical overhauls. On removal, the teeth were putrescent. Medical measures, which would have otherwise failed, led to arthritic recovery in a few weeks and the individual works, with restored health, as a storeman.

In dissociating myself from statistical extremists, who have attributed even such remote phenomena as myomata to focal sepsis, I would like to affirm as a result of unremitting association with this field, that the hypothesis of focal infection is not erroneous, though its subtlety often defies analysis. May I be permitted to state some working rules?

The term "focal infection", which infers a faculty for surgical elimination, should be dropped in favour of some such term as "instrusive homologous toxæmia". This distinction is necessary because many diffuse infections of non-surgical or medical type clamour for recognition in our ætiological and therapeutic programme.

The removal of an obvious, seemingly fool-proof focus does not always mean the removal or treatment of the responsible and often unobtrusive focus.

A primary homologous focus often leads to a general reduction of resistance with invasion at other sites. So we find chronic bronchitis as a reflex of antral and tonsillar sepsis, chronic furuncular disorders in association with latent abscesses of apical dental type, and gall-bladder pathology revealed at autopsy, in one-third of all individuals over fifty (Mayo Clinic), presumably in individuals with a defective alimentary habitus.

Homologous infection may include: chronic otitis (with or without discharge), mucosal thickening or polypi of the accessory nasal sinuses (the sole subjective symptom may be some nasal obstruction and catarrh), necrosis of the bone following trauma of the olfactory region, pyorrhœa, all devitalized teeth whatever the X-ray estimation, crowned teeth, pivots, chronic and painless tonsillitis associated with adenitis and glandular tenderness, chronic bronchitis and bronchiectasis, cholecystitis, diverticulitis, chronic constipation especially with impacted fæcal deposits, mucous colitis, obstructive appendicitis associated with alimentary stasis, chronic pyelitis or cystitis, ulceration of the cervix, post-venereal prostatitis, varicose ulcers, anal fistula, recurrent furuncles (occasionally, but not always noxious), and long-standing suppurative ingrowing toe-nails. It can be stated as axiomatic that if a victim of toxic absorption has had a single focus surgically eliminated, yet harbours one or more of the foregoing conditions, focal infection cannot be claimed to have been dealt with. There is no surer test of the existence of a still operative focus than an exaggerated hypersensitivity of the individual to vaccine therapy, as is obvious in the subacute stages of an infective arthritis.

Regarding special situations, it cannot be over-emphasized that no devitalized tooth, however negative the X-ray report, fails, on culture, to produce a bacterial growth, usually streptococcus of viridans or hæmolytic type. In attempts to turn defeat into victory, the term "depulped" teeth (credited with vital dentine) has come into vogue. Here the important and realistic fact is the fragillated, periodontal membrane which operates as a necrotic bridge of transport between the buccal bacterial antigens and the general tissues of the host.

It is to be noted that the appendix has been included as a debatable member in the team of toxic focal agents. In all cases where appendicectomy had been ultimately performed in cases of irremedial alimentary stasis and dyspepsia, hæmolytic colon bacilli and/or streptococci therefrom have without exception possessed a high specificity in vaccine therapy, directed to arthritic lesions. In fact after a held-up 50% recovery derived from elimination of a dental abscess, the balance of the trouble has cleared up after recourse to vaccine therapy derived from an appendical source.

Treatment of a chronic bronchitis as a background to chronic spondylitis or arthritis ranks equally with or transcends in importance special measures directed to the secondary rheumatic condition. Here rheumatism (labourer's spine) is a disease of a disease, affecting an over-used toxically spoiled, musculo-skeletal system. Actual histories show that arthritis may develop after an accident or as the result of trauma in individuals who have, earlier, been the subject of tissue spoiling from prolonged osteomyelitis or peritoneal suppuration.

Thus a robust nurse of athletic type, who had been brought up as a horse-woman on a station and had spent six months in hospital with a suppurating appendix, later developed rheumatoid arthritis as the result of the trauma entailed in lifting a heavy invalid. She was definitely not the type to succumb spontaneously to this disorder, and returned to duty in one month with vaccine therapy that took into account the alimentary source of her toxæmia. Dispersal of inflammatory tenderness in the region of adhesions and of an associated dyspepsia contributed to this end.

It is thus reasonable to point out to Dr. Shallard and his adherents that if the elimination of obvious dental or tonsillar sepsis or other common types of focal infection fails to lead to more than fractional or temporary relief, the ætiological hypothesis regarding focal infection cannot be condemned unless the existence of the other factors in the above catalogue can be discounted.

As a consequence of widely held doubts regarding the validity of the concept of focal sepsis, vaccine therapy (never to be regarded as more than a fractional arm in treatment) has been unavoidably discredited as a therapeutic offshoot of the theory of focal infection.

Obviously the key must fit the lock; and before we condemn what is a valuable weapon, when used with discrimination, it is necessary to realize that a vaccine which is specifically adapted to an arthritic sufferer with chronic bronchitis, will not be specific in a case in which the primary toxic source is cervicitis, pyelitis or mucous colitis. We may yet learn that an additive synergism born of a cross-fire from multiple foci may be a significant factor in the attainment of a threshold of absorption of a degree which can lead to toxic spoilation.

All rheumatologists deplore the fact that some 5% to 10% of arthritics appear to fail to respond to any therapy. These may be in the class of Mendelian recessives, as in the case of some tubercular subjects, where immunity-providing genes are concerned.

As it is at times impossible to identify the part played by an array of foci, or obtain a genuine autogenous vaccine, a stock vaccine which includes as a basis, diverse streptococci (hæmolytic and viridans), with adjunctive additions of micrococci, coryzal organisms and of the flora of the gut, such as colon bacilli (hæmolytic and non-hæmolytic) and enterococci, is of definite value in small dosage as an empirical concession to the imponderables of focal infection.

The question also needs to be put, when and to what degree can we expect spontaneous recovery as a result of elimination of responsible homologous infection? It is necessary in this connexion to realise that the tissue sequelæ to the long-range effects of toxic spoilation (due in part to lytic effects of the antibacterial measures of the host) should be classified as of first and second degree.

If the metabolic disorder is of short range or first degree type and purely biochemical, being confined to the floating metabolism of the tissue cells, spontaneous recovery may occur on removal of the infective agent. If toxic spoilation has led in turn to the second degree disorders of structural and trophic type seen as chronic fibrositic change, or obvious tissue deformity, or again if endocrine and climacteric metabolism is deranged, many accessory measures over and above identification and eradication of a focus will be required to restore the *status quo*.

In the hyperkatabolism of atrophic arthritis, the carbohydrates, fat and protein of the host do not appear to be insulated against lytic agents which are directed by the immunity process against the carbohydrate, protein and fats, of which bacterial antigens are composed. In this lysis, the individual is seemingly the trophic victim of his own immunity "barrage".

Vaccine therapy used in subreactive or mildly reactive doses appears to transform this into a process of more limited, "sniping" type.

This lytic hypothesis which gained approval of authorities on the Empire Council of Rheumatism may well explain the phenomenon of fibrositis.

Between parenchymatous muscle and supporting fibrous cells exists a local, trophic equilibrium of interchange in which the tissue contents of muscle cell appear to be converted to those of the fibrous and fatty cell, in lapses which ensue between periods of training, just as the converse appears to be the case on resumption of athletics. Artificial lysis of the type earlier noted and due to immunity excess, appears to give rise to the muscle cell exudate which is characteristic of the preorganized stage of fibrotic hypertrophy, and later evokes fibrous mitosis and cell formation.

A legacy from the present stress of industry and war will be the wear and tear or trophic diseases of civilization. It would be well if our profession be ready in the post-war period to assemble available knowledge and found organized clinics of the type so ably conducted overseas before the war by the British Empire Council of Rheumatism.

Yours, etc.,

FRANK TRINCA.

111, Collins Street,
Melbourne,
August 19, 1942.

Naval, Military and Air Force.

CASUALTIES.

ACCORDING to the casualty list received on September 14, 1942, Captain R. G. Lyne, A.A.M.C., Rose Bay, New South Wales, is reported to have died of wounds.

Obituary.

CECIL TANKO.

We regret to announce the death of Dr. Cecil Tanko, which occurred on August 30, 1942, at Adelaide, South Australia.

ERIC MARTIN HALL.

We regret to announce the death of Dr. Eric Martin Hall, which occurred on September 3, 1942, at East Malvern, Victoria.

CONSTANCE ELLIS.

We regret to announce the death of Dr. Constance Ellis, which occurred on September 10, 1942, at South Yarra, Victoria.

Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Ralston, Frederick George, M.B., B.S., 1899 (Univ. Sydney), c.o. McIlwraith McEacharn Limited, 19, Bridge Street, Sydney.

Thomas, David Lloyd, M.B., B.S., 1942 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Edye, John Andrew, M.B., B.S., 1942 (Univ. Sydney), 13, Kirkoswald Avenue, Mosman.

Hall-Johnston, Doreen Olive (Moore), M.B., B.S., 1938 (Univ. Sydney), "Tannabah", Gilbert Park, Manly.

Chesher, Rupert George, M.B., 1941 (Univ. Sydney), District Hospital, Parramatta.

Allen, Loraine Georgiana, M.B., B.S., 1942 (Univ. Sydney), District Hospital, Bathurst.

Monk, Ian, M.B., 1940 (Univ. Sydney), 37, Raglan Street, Mosman.

Sterling-Levis, Miles, M.B., B.S., 1937 (Univ. Sydney), c.o. Bank of New South Wales, Head Office, 341, George Street, Sydney.

Kendall, Richard Bruce, M.B., B.S., 1942 (Univ. Sydney), Balmain District Hospital, Balmain.

Halliday, Francis Bathurst, M.B., B.S., 1940 (Univ. Sydney), 28, Burns Road, Wahroonga.

Rosenfeld, Israel (registered in accordance with the provisions of Section 17A of the *Medical Practitioners Act*, 1938-1939), District Hospital, Goulburn.

Waugh, Isobel Patricia, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Tyrer, John William Howard, M.B., B.S., 1942 (Univ. Sydney), 227, Old South Head Road, Bondi.

Pullen, Wallace James, M.B., B.S., 1939 (Univ. Sydney), N27148, Captain W. J. Pullen, Regimental Medical Officer, 36th Battalion, Australian Military Forces, Australia.

The undermentioned have been elected members of the South Australian Branch of the British Medical Association:

Sheppard, Mark Yeatman, M.B., B.S., 1942 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Kerr, Challen Sydney, M.B., B.S., 1942 (Univ. Adelaide), B.Sc., 1934 (Univ. Springfield, United States of America), South Esplanade, Semaphore.

Medical Appointments.

Dr. Harold Newton Zimmer and Dr. Wilfred Ewart Macmillen have been appointed Public Vaccinators for Victoria.

Books Received.

"Diseases of Women", by ten teachers under the direction of Clifford White, M.D., B.S. (London), F.R.C.P. (London), F.R.C.S. (England), F.R.C.O.G., edited by Sir Comyns Berkeley, Clifford White and Frank Cook; Seventh Edition; 1942. London: Edward Arnold and Company. Demy 8vo, pp. 444, with 168 illustrations. Price: 18s. net.

Diary for the Month.

SEPT. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 24.—New South Wales Branch, B.M.A.: Branch.
SEPT. 24.—South Australian Branch, B.M.A.: Branch.
SEPT. 25.—Federal Council of the B.M.A. in Australia: Meeting in Melbourne.
SEPT. 25.—Queensland Branch, B.M.A.: Council.
SEPT. 25.—Tasmanian Branch, B.M.A.: Council.
OCT. 1.—South Australian Branch, B.M.A.: Council.
OCT. 2.—Queensland Branch, B.M.A.: Branch.
OCT. 6.—New South Wales Branch, B.M.A.: Council Quarterly.
OCT. 7.—Western Australian Branch, B.M.A.: Council.
OCT. 9.—Queensland Branch, B.M.A.: Council.
OCT. 13.—Tasmanian Branch, B.M.A.: Branch.
OCT. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee, Organization and Science Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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